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THE MANAGEMENT OF ACUTE UPPER GASTROINTESTINAL HEMORRHAGE*

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The management of acute upper gastrointestinal hemorrhage continues to challenge the ingenuity and interest of all who have occasion to deal with this problem. The last decade has seen considerable progress and the wide differences of opinion which formerly characterized the treatment of this condition are beginning to crystallize. The present paper is intended to depict the general trends which are becoming evident in the management of this problem throughout this country.

ETIOLOGY

The etiology of acute massive upper gastrointestinal hemorrhage is summarized in table I which lists the dominant causes in 95 per cent of cases. There always will remain an occasional bizarre cause of severe hemorrhage which will only be recognized at operation or autopsy. Among these are such conditions as rupture of an aneurysm invading the esophagus, carcinoma of the esophagus invading the aorta, bleeding from arteriosclerotic vessels in diverticula or the hemorrhage associated with spontaneous rupture of the esophagus. In connection with this last condition it is of interest that massive upper gastrointestinal bleeding may be a prominent feature of so-called spontaneous rupture of the esophagus and may in some instances precede the development of the severe pain which usually characterizes this condition.

It can be seen from table I that the vast majority of conditions which occasion

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TABLE I

Etiology of acute upper gastrointestinal hemorrhage in 95% of cases

Author	Duodenal Ulcer	Gastric Ulcer	Jejunal Ulcer	Gastric Neoplasm	Gastritis	Esophageal Varices
Allen[1]	94	42	8	48	2	37
Costello[6]	171	33	4	4	42	24
Warthin[16]	48	7	1		6	8
Welch[17]	210	41	8	8	20	62
Peter Bent Brigham Hospital	127	15	6	5	17	14
	650	138	27	65	87	145
	58%	12%	2%	6%	8%	13%

massive upper gastrointestinal bleeding may be benefitted by surgical intervention under appropriate circumstances. Even in gastritis there at times may be superficial erosions which will continue to bleed despite medical management.

The massive hemorrhage which frequently characterizes esophageal varices is not infrequently fatal and, because of this, increasing efforts to control bleeding from this source by direct attack are being made. Packing of the esophagus or compression by an inflatable bag are among the measures which temporarily will arrest massive bleeding. When bleeding recurs following release of compression, surgical intervention with direct suture of the varices may be indicated.[10] Our own experience with this procedure has been disappointing. A similar experience has characterized the work of Warren and Warthin at the West Roxbury Veterans Administration Hospital where in recent years a more vigorous surgical attack has been made in cases of exsanguinating hemorrhage from esophageal varices.[14] The results have been disappointing in that in a number of instances, although the hemorrhage has been controlled, death has ensued from hepatic coma. In our experience (at the Peter Bent Brigham Hospital), on release of the compression if bleeding recurs, it has not been possible to control it subsequently. It appears that a much more vigorous attack must be made early in the course of massive bleeding from esophageal varices if surgical intervention is to be successful. A routine comparable to that employed in the management of bleeding ulcers may establish more precise indications for immediate surgical intervention. Warren now believes that, if bleeding is sufficiently severe to require temporary control, immediate measures for emergency transesophageal ligation of the varices should be taken. Contraindications are intense jaundice and severe hepatic failure.[14]

The major causes of acute upper gastrointestinal hemorrhage remain duodenal ulcer, gastric ulcer and jejunal ulcerations. Indeed, as can be seen from table I, these causes together with gastric neoplasm account for over 80 per cent of all cases of acute massive upper gastrointestinal hemorrhage and it is to a consideration of the management of bleeding duodenal and gastric ulcers that this paper is principally directed.

Mention must be made, however, of the patients in whom no source of bleeding

TABLE II

Massive gastric hemorrhage of obscure etiology

No abnormality demonstrable by roentgenography or fluoroscopy

100 cases

Age: 18–40 years	21 cases (21%)
40–75 years	79 cases (79%)
Cause of Bleeding	
Hemorrhagic erosions	66 cases 66%
Hypertrophic gastritis	22 cases 22%
Gastric ulcer	2 cases 2%
Malignancy	2 cases 2%
Unexplained	8 cases 8%

can be found. Table II lists the etiologic factors in a series of cases analyzed by Gray.[8] In these instances, if the bleeding subsides under management, a careful investigation as to the exact site of the bleeding may be undertaken. At times the use of the string test and of intestinal intubation are helpful in locating an obscure source of bleeding which cannot be detected by the roentogenogram or gastroscopy. The importance of early roentgenograms in obscure cases has been emphasized by Zamcheck[18] and his associates at the Boston City Hospital. Cooper and Ferguson[5] have resorted to blind subtotal gastrectomy in a series of 18 patients in whom no evidence of a source of hemorrhage could be established preoperatively. In 6 of these patients at operation no source was identified by exploration, but in each instance a resectable mucosal erosion or acute ulceration was established as a source of bleeding after gastrectomy had been performed.

THE PROBLEM OF DIAGNOSIS

The problem of diagosis as to the etiology of upper gastrointestinal hemorrhage is a formidable one particularly in relation to the small group of patients mentioned previously in whom no source of bleeding can be readily identified by the roentgenogram or gastroscopy. The use of early roentgenograms in establishing the diagnosis in acute massive upper gastrointestinal hemorrhage is gaining support as a result of the excellent studies of Zamcheck[19] and Warthin.[15] At the Peter Bent Brigham Hospital we have not resorted to immediate roentgenographic examination in every patient. It has been our belief that, in a majority of patients, a substantial clue as to the nature of the bleeding is obtained from the history, physical findings and laboratory data. Under these circumstances we prefer to treat the patient with a presumptive diagnosis and reserve roentgenographic study only for selected patients in whom there is a real doubt as to the etiology. Formerly we thought that roentgenograms should not be employed during the bleeding phase and we undertook the examination only in the most severe cases as a step prior to surgical intervention. This has not proved satisfactory and we now prefer, if there is real doubt as to the nature of the bleeding, to make roentgenographic studies shortly after the circulation has

been stabilized. One of the advantages of this is the hope that small, acute ulcerations which frequently heal rapidly and are overlooked when roentgenographic examinations are made two or three weeks after the bleeding has subsided may be detected in the acute phase. The experiences of Zamcheck and Warthin indicate that this is the case. In some centers cirrhosis with associated varices constitute a much larger percentage of cases than others. Where this is true, as in large city hospitals, additional studies may be required to establish a diagnosis of an associated ulcer or to exclude varices in the presence of an ulcer. Along these lines the use of the bromsulfalein disodium retention test has been shown to be of value by Zamcheck and his associates. Despite all such measures, however, there always will be a small percentage of patients in whom the diagnosis remains obscure and in whom, if the hemorrhage continues massively, surgical intervention must be undertaken. We now believe strongly that when there is exsanguinating hemorrhage, the cause of which is not clear, emergency esophagoscopy should be done. If varices are found, an emergency transthoracic ligation of the varices can be carried out. If no varices are found, laparotomy should be done. If no cause of bleeding is found, a *blind gastrectomy* may be required. Our own experience and that of others indicate that this will salvage an occasional patient who otherwise would be lost.

THE CHOICE OF MANAGEMENT IN SEVERE HEMORRHAGE FROM GASTRIC OR DUODENAL ULCER

Although the controversy which has existed for generations over the proper treatment of bleeding gastric or duodenal ulcer continues, there is a growing trend towards the adoption of surgical intervention in selected cases. There still remains a few who would resort to medical management in every case, accepting the death of a few patients as inevitable. The work of Stewart[13] indicates that good results can be obtained by prompt surgical intervention in every case. We have been opposed to both of these choices of management. The first on the grounds that it condemned a few patients needlessly to death and the second on the grounds that it forced the surgeon to operate upon many patients who might not require surgery, and it also forced him to operate upon a large number of patients under circumstances which were not wholly favorable. Two factors contribute to the unfavorableness of surgical intervention in the acute phase. First, despite the great advances in anesthesia and blood replacement, there is a pressure associated with operation when it is done on a patient who is or has been in shock which may not always allow the most definitive procedure to be performed. Second, from the pathologic point of view, the acutely bleeding ulcer is an active ulcer. An active ulcer, with the associated edema and inflammation, is the principal cause of surgical mortality in relation to leakage from the duodenal stump. For these reasons we have always preferred to undertake surgery in the selected patients in whom it appears that the bleeding cannot be stopped by conservative means.

Since 1946 it has been the policy at the Peter Bent Brigham Hospital to treat all patients with bleeding ulcer conservatively, but to utilize surgical

intervention promptly in selected cases. The problem of selection will always remain a difficult one, but since our initial emphasis on the importance of the rate of bleeding as assessed by the amount of blood required to stabilize the circulation, similar approaches have been utilized by Warthin and Warren,[16] Bowers,[4] Alsobrook,[2] Mayo[11] and Saltzstein.[12] The composite mortality from all of these groups now is well below an over-all mortality rate of 5 per cent, a highly creditable figure. Combined medical and surgical management is the essence of successful treatment.

THE MEDICAL MANAGEMENT OF ACUTE GASTROINTESTINAL BLEEDING

Every patient on admission to the hospital must be seen by representatives of both the medical and the surgical services. The patient is kept on rigid rest in bed and a chart of the pulse, respiration and blood pressure is maintained at intervals of 15 to 20 minutes. If the patient is in shock, immediate transfusion is given and an estimate of the severity of the bleeding is made on the basis of the amount of blood required to stabilize the circulation. In the vast majority of patients 500 to 1000 cc. of blood is sufficient to bring about a prompt stabilization of the pulse and blood pressure. If more than 2000 cc. of blood is required to stabilize the circulation, it is our belief that the patient is continuing to bleed so massively that immediate and prompt surgical intervention is required. Under these circumstances as soon as the circulation has been stabilized by multiple transfusions, operation is undertaken if there is a clear-cut indication as to the site of bleeding. If this is not evident or probable, the patient is studied radiographically following which procedure surgical intervention is undertaken. We do not believe that a negative roentgenogram makes it unwise to operate since minute ulcerations may be found, but we do believe when there is no clue as to the diagnosis, that an emergency roentgenogram may disclose evidence of exceedingly great value which will facilitate the performance of the operation. Esophagoscopy, as previously mentioned, may be required to exclude varices.

Assuming that the circulation stabilizes promptly with one or two transfusions, the following program is instituted. At the time of the first venipuncture for transfusion blood is taken for the following laboratory tests: the usual complete blood examination, blood typing and cross matching, a bleeding and clotting time, a prothrombin time, a plasma protein, a blood urea nitrogen and serum chloride, potassium, sodium and carbon dioxide content. All stools which the patient passes are examined for occult blood.

The key to the management of the acute case is blood replacement. One is guided by the response to transfusion. In the early stages blood is given very rapidly, but once the circulation is stabilized, additional transfusions are given slowly not to exceed a rate of 500 cc. in two to four hours. In these acute cases the hematocrit is of comparatively little value. If it is low initially, it merely indicates that the patient has been losing blood slowly and has had a chance to dilute his circulation prior to the final episode of bleeding which initiated circulatory collapse. This combination of anemia and hypovolemia can be treated initially by whole blood transfusion, but infusions of packed red cells may be

required to raise the hematocrit to normal levels. Excessive whole blood transfusions given because the hematocrit is still below normal may overload the circulation and produce pulmonary edema. In the very acute cases the hematocrit usually is not far below the normal range initially and falls over a period of 24 hours. During this phase of therapy the best guides to transfusion are the clinical signs of syncope, sweating, fainting and weakness and the changes in the pulse and the blood pressure. The pulse rate should be kept in the neighborhood of 100 or below and the blood pressure above a level of 100/80. There are, of course, individual variations which must be weighed in the balance, such as the size of the patient and the presence or absence of known hypertension. In occasional cases we have done blood volume studies by means of radioactive chromate, but we have not undertaken this as a specific adjunct to clinical management.

If, as is the case 90 per cent of the time, the circulation stabilizes effectively with replacement therapy, the key to continued successful management is effective neutralization of gastric acidity. Frequent feedings of milk, cream, and antacids, or the institution of a modified Meulengracht regimen are essential. Milk and cream and an antacid are entirely satisfactory. If nausea or vomiting ensues, cream should be eliminated and the quantity of milk reduced to not more than 30 cc. an hour. It can be gradually increased. It has been our experience that if repeated vomiting occurs under these circumstances, it is due to continued bleeding and one is faced with one of the great danger signals, namely, recurrent hematemesis. Under these circumstances the need for surgical intervention becomes likely.

Morphine and other narcotics are not employed. Sedation is obtained by means of sodium phenobarbital in small amounts at intervals of four to six hours. Atropine to reduce gastric acidity is given regularly in small doses at four hour intervals. Vitamin C and vitamin K are administered to all patients.

If the patient is able to take the above regimen, his nutrition is well maintained and supplementary feedings are not necessary. If 100 cc of milk and cream are given every hour during the day and at two hour intervals during the night, a total of 2400 cc. of fluid is given and the amount of calories ranges up to 3000. There is a liberal quantity of protein in this intake and this is further supplemented by the indicated transfusions.

Under this regimen the vast majority of patients will stop bleeding within 24 to 48 hours. Occasionally mild, recurrent episodes of hemorrhage may occur, but in our experience no patient who has been carried longer than five days on this program and in whom all signs of bleeding have subsided, has subsequently developed massive hemorrhage requiring surgical intervention. The indications for surgery are not precise, however, and considerable judgment must be exercised in weighing them.

THE INDICATIONS FOR SURGERY

A great variety of signs have been used as indications for surgical intervention. Emphasis has been placed upon the age of the patient, the response to conserva-

tive management, the number of previous hemorrhages and the inadvisability of delay if hemorrhage does not subside promptly. Our belief is that the rate of bleeding still remains the dominant factor. We have divided the various factors into three groups as guides in following these patients.

Group A—*Danger Signals.* These include repeated nausea and hematemesis, persistent severe pain, recurrence of bleeding on a good medical regimen, and recurrent fainting, sweating and weakness. Any of these factors place the patient in a category in which surgical intervention becomes probable. Under such circumstances all facilities are secured so that prompt operative intervention may be undertaken. We do not believe that any of these factors taken alone or together constitute an absolute indication for operation unless there is circulatory collapse which fails to respond to transfusion. These factors are grounds, however, for emergency diagnostic measures if the probable cause of the hemorrhage is not known.

Group B—*Contributory Factors.* These include the age of the patient, the location of the ulcer if known, the significance of delay in operation, the number of previous hemorrhages and accurate information regarding associated diseases or other possible causes of hemorrhage. These are factors which in themselves do not indicate the need for operation, but which influence the surgeon in electing to undertake it or withhold it. In our experience age is significant in that the older the patient, the more likely the hemorrhage is to be massive. However, young patients may have exsanguinating hemorrhages and may die if surgery is not undertaken. Contrariwise, the older patient as a rule is a less suitable candidate for extensive emergency surgery. No hard and fast rules can be established with reference to age. It is a factor which must be balanced by the surgeon in the individual case.

The location of the ulcer, if it is known, is of great significance. By and large in our experience gastric ulcers have bled more massively than duodenal ulcers and they are approached more readily surgically. For this reason, if it is known that the patient is bleeding from a gastric ulcer, we employ surgery with less hesitation than in a duodenal ulcer under comparable circumstances.

A great deal of emphasis has been placed upon the need to operate within 48 hours of the onset of bleeding. This is not necessary, however, since we know that delay is of importance only if it is delay in a patient whose circulation is unstable. Many patients may bleed moderately for several days and then respond very effectively to medical management. In other instances moderately severe bleeding may become massive after a period of several days and can be controlled only by surgical intervention. We have operated upon a number of patients after 48 hours of treatment in the hospital and there have been no deaths under these circumstances. On the other hand, we believe very strongly that if the circulation cannot be readily and substantially stabilized by transfusion, immediate operative intervention is necessary. Delay with a patient in shock is fatal.

We have not considered that in the individual the number of previous hemorrhages is significant. In general, if the patient has bled many times before

and has ceased spontaneously, the probability is that he will do so again. We do not believe, however, that this is solid ground to rest upon and, whether or not the patient is in his first or fifth hemorrhage, our program is identical and we utilize the same indications for surgical intervention.

An accurate diagnosis and an awareness of associated diseases merely supply factors which the surgeon must weigh in the balance. Obviously, if a patient is in excellent condition and one knows exactly where the site of bleeding is, operation may be undertaken with less hesitation than in a very poor-risk patient in whom the site of bleeding is unknown and in whom a prolonged operation might in itself prove fatal. Here again, these are factors for which there are no rules and every surgeon must exercise his own best judgment.

Group C—*The Definitive Indication.* The definitive indication for surgery is the rate of bleeding as manifested by the response to transfusions. In a general way, as outlined in earlier papers,[7, 9] if the circulation cannot be stabilized initially by 2000 cc. of blood, it indicates that the patient has had a massive hemorrhage and is continuing to bleed massively. Under these circumstances blood is given very rapidly and as soon as the circulation is stabilized and all permissible measures that can be taken to establish the diagnosis have been done, operation must be undertaken. In those patients who stabilize immediately following transfusions, if it is necessary to continue transfusion at the rate of more than 1000 to 1500 cc. daily, it is unlikely that the patient will respond to conservative measures and prompt operation should be undertaken. In the vast majority of patients the decision as to whether surgery will be necessary can be made within 12 to 24 hours. Occasionally patients will continue to bleed at a rate which can just about be matched by transfusions of 500 cc. every eight hours. In the past we have tended to continue with such patients, hoping that stabilization would ensue ultimately. It is gratifying that no patients who have been carried in this fashion for more than five days have died, but emergency operation has been necessary as late as nine days and in one instance as late as 30 days following admission to the hospital. For that reason we believe strongly now that if the circulation is stabilized but bleeding continues for more than three days, operation should be undertaken. Under such circumstances we have in one instance encountered a gastric carcinoma which was the source of bleeding. As a result of our experience we now feel quite confident that we can recognize the more severe bleeders and bring them to surgery within 48 hours of admission to the hospital.

SURGICAL TECHNIC

Once the indications for surgery are clear-cut, certain specific measures must be undertaken. While the patient is on medical management gastric tubes are never used, but the moment surgery is elected gastric lavage is done and the stomach emptied. Prior to the induction of anesthesia two intravenous drips are established; one of which is located in a cannulated vein to insure that continuous infusion can be maintained. A catheter is inserted in the bladder and a record is made of the hourly output of urine. As anesthesia is induced the

transfusions are stepped up sufficiently to maintain an adequate circulation, particularly with reference to blood pressure.

The details of the surgical management of acute hemorrhage are not within the scope of this paper but several points warrant emphasis. Adequate exposure must be obtained. This is no time for small or limited incisions. In many instances the site of the bleeding may be evident on exploration and palpation. If this is not the case, mobilization and transection of the stomach at any convenient point just above the pylorus is undertaken as shown in figure A. The divided stump then is opened as indicated and the site of bleeding determined by direct inspection. If a duodenal ulcer is encountered and there is active bleeding, simple transfixion of the ulcer bed to control active hemorrhage is undertaken. Care must be exercised if the ulcer lies in the second portion of the duodenum lest the transfixing suture injure the common duct. The purpose of this suture

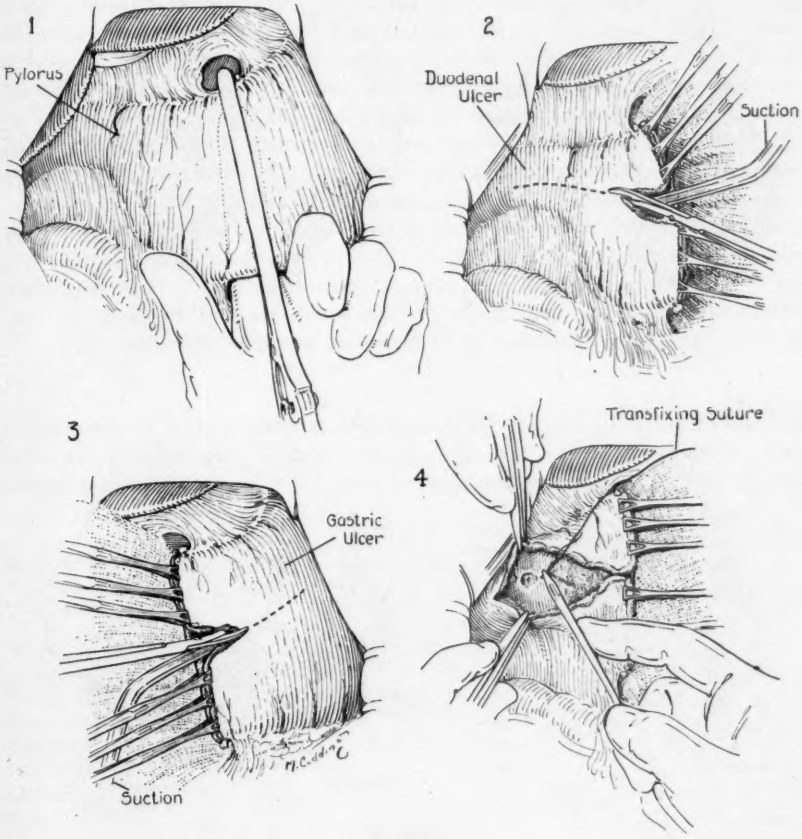

FIG. A

merely is to control active hemorrhage from the ulcer bed. If no site of bleeding is found in the distal portion of the stomach, the proximal section of the stomach also is opened and inspected directly as shown in figure A-3. It may be necessary to evert portions of the stomach through this large opening in order to be certain that minute but serious mucosal ulcerations are not overlooked.

At times it may be difficult to reach the ulcer itself or to mobilize the duodenum, and under those circumstances the lateral peritoneal reflexion over the duodenum should be divided and the duodenum and head of the pancreas mobilized sufficiently to bring it well up into the wound for adequate manipulation and inspection. Ideally, the ulcer should be excised as shown in figure B, but if it lies well distally or if there are technical difficulties encountered, we believe it permissible to exclude it as shown.

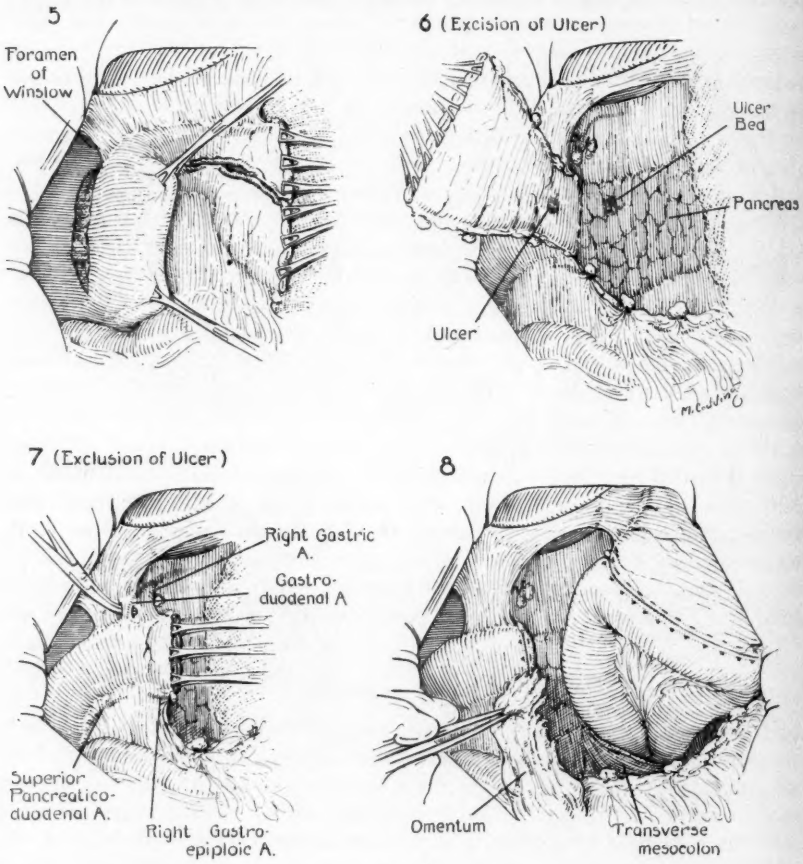

FIG. B

Under these circumstances, namely, a patient who is being operated upon for an exsanguinating hemorrhage, definitive cure of the ulcer is not the primary objective. Consequently, additional procedures such as vagotomy should not be undertaken. The extent of the gastric resection must be determined in part by the reaction of the patient to the procedure. Under most circumstances the standard ⅔ to ¾ gastric resection may be undertaken, but in certain instances, particularly in the elderly or poor-risk patient, a lesser procedure must of necessity be done. If this should prove inadequate subsequently, additional measures may be considered as elective procedures later. The primary purpose of the emergency operation is to control bleeding and to save the life of the patient. Fortunately, as previously mentioned, in a majority of instances, as a consequence of adequate blood replacement and good anesthesia, a standard gastric resection can be done.

The management of the duodenal stump may pose a serious problem, particularly in instances where excision of the ulcer has been undertaken and after completion of the procedure it is found impossible to get adequate closure because of edema and the shortness of the remaining stump. Under these circumstances, rather than attempt further mobilization of the duodenum and possible injury to the common duct, a catheter duodenostomy and a jejunostomy as recommended by Welch should be made and under these circumstances, if there is any loss of fluid from the duodenal stump, it can readily be introduced through the jejunostomy tube and there is no interference with the patient's nutrition. Complete control of the stump is obtained; there is no danger of perforation or leakage and the small duodenal fistula which ultimately will result in the majority of patients closes promptly. When no source of hemorrhage is found in the stomach and there is no evidence of blood in the stomach, minute attention should be directed to the upper small intestine. Vascular anomalies, carcinoma and jejunal diverticula are rare but important causes of severe upper gastrointestinal bleeding.

RESULTS

The results of the management of acute hemorrhage from gastric and duodenal ulcer in a collected series of cases including our own is shown in table III. The over-all mortality rate as can be seen is well below 5 per cent. In each of the

TABLE III

Results of combined medical and surgical management of bleeding gastric and duodenal ulcer

	Cases	Deaths	% Mortality	No. of Surgical Cases
Dunphy, Hoerr (1948)	45	1	2.2%	4
Warthin (1949)[16]	56	2	3.6%	9
Bowers (1950)[4]	150	2	1.3%	2
Peter Bent Brigham Hospital (1951)	127	2	1.5%	15
Alsobrook[2]	87	4	4%	12
Saltzstein (1953)[12]	343	18	5%	68
Mayo (1954)[11]	99	5	5%	23

series quoted the emphasis was placed on a combined medical and surgical approach, operation being employed only in the worst cases. Differences in the number of patients subjected to emergency operation may be accounted for by chance since some of the series are small, by an actual difference in the type of patient encountered and by variations of interpretation. It is most important that patients no longer be permitted to die without the benefit of surgical intervention. Ideally, the medical mortality rate should be zero; the surgical mortality rate below 10 per cent and the over-all mortality rate well below 5 per cent.

It is evident that this ideal is being approached and, as Bowers has said recently, there seems at last to be a general meeting of minds on this important problem.[3]

REFERENCES

1. Allen, A. W.: Acute massive hemorrhage from upper gastrointestinal tract, with special reference to peptic ulcer, Surgery *2:* 713 (Nov.) 1937.
2. Alsobrook, W. L., Schell, M. W., and McCleery, R. S.: Combined service approach in treatment of bleeding peptic ulcer, Gastroenterology *21:* 71 (May) 1952.
3. Bowers, R. F.: Discussion of Mayo's paper before the Society of University Surgeons, (Feb.) 1954.
4. Bowers, R. F., and Rossett, N. E.: Bleeding peptic ulcer; favorable results by conservative treatment, Ann. Surg. *132:* 690 (Oct.) 1950.
5. Cooper, D. R., and Ferguson, L. K.: Gastric resection for upper gastrointestinal hemorrhage of undetermined cause, J. A. M. A. *151:* 879 (March) 1953.
6. Costello, C.: Massive hematemasis, Ann. Surg. *129:* 289 (March) 1949.
7. Dunphy, J. E., and Hoerr, S. O.: Indication for emergency operation in severe hemorrhage from gastric and duodenal ulcer, Surgery *24:* 231 (Aug.) 1948.
8. Gray, S. J.: Personal communication.
9. Hoerr, S. O., Dunphy, J. E., and Gray, S. J.: Place of surgery in emergency treatment of acute massive upper gastrointestinal hemorrhage, Surg., Gynec. & Obst. *87:* 338 (Sept.) 1948.
10. Linton, R. R., and Warren, R.: Emergency treatment of massive bleeding from esophageal varices by transesophageal suture of these vessels at time of acute hemorrhage, Surgery *33:* 243 (Feb.) 1953.
11. Mayo, H. W., Jr., and Jennings, K. O.: Experiences with management of severe bleeding from peptic ulcer, Surgery (to be published)
12. Saltzstein, H. C., Mahlin, M. S., and Scheinberg, S. R.: Bleeding peptic ulcer, A. M. A. Arch. Surg. *67:* 29 (July) 1953.
13. Stewart, J. D., Schaer, S. M., Potter, W. H., and Massover, J. A.: Management of massively bleeding peptic ulcer, Ann. Surg. *128:* 791 (Oct.) 1948.
14. Warren, R., and Warthin, T. A.: Personal communication.
15. Warthin, T. A., Ross, F. P., Baker, D. V., Jr., and Wissing, E.: Management of upper gastrointestinal hemorrhage, Ann. Int. Med. *39:* 241 (Aug.) 1953.
16. Warthin, T. A., Warren, R., and Wissing, E. G.: Combined medical and surgical management of upper gastrointestinal hemorrhage, New England J. Med. *241:* 473 (Sept. 29) 1949.
17. Welch, C. E.: Treatment of acute, massive gastroduodenal hemorrhage, J. A. M. A. *141:* 1113 (Dec. 17) 1949.
18. Zamcheck, N., Chalmers, T. C., Ritvo, M., and Osborne, M. P.: Early diagnosis in massive gastrointestinal hemorrhage, J. A. M. A. *148:* 504 (Feb. 16) 1952.
19. Zamcheck, N., Chalmers, T. C., White, F. W., and Davidson, C. S.: Bromosulphalein test in early diagnosis of liver disease in gross upper gastrointestinal hemorrhage, Gastroenterology *14:* 343 (March) 1950.

THE RECOGNITION AND MANAGEMENT OF PERIPHERAL VASCULAR DISORDERS*

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The problems associated with peripheral vascular disorders touch almost every field of medicine and surgery, so much of the confusion and perhaps much of the misunderstanding about these stepchildren of surgery may stem from the fact that we have tried to treat them as isolated clinical entities rather than disturbances of vascular function.

It is true that the fundamental nature of most of the peripheral vascular disorders still is very poorly understood, so it becomes even more important to try to overcome the ischemia in an extremity, and thus prevent necrosis of the soft tissues, rather than try to eliminate the cause or eradicate the underlying pathologic process. Permanent recovery usually depends upon the early recognition of the disturbed function with institution of definitive treatment before serious complications have made their appearance.

The field of peripheral vascular disorders is a very broad one and most of the disturbances present both medical and surgical aspects. Because of the limited time available, we shall discuss only those disturbances of circulation in the extremities which are of special interest to us as surgeons, and we shall assume then that the cardiac function and great blood vessels of the chest and abdomen are relatively normal.

The recent widespread interest in the pathologic-physiology of vascular disorders has yielded information which gives us all a much clearer concept of the mode of onset of changes in the peripheral distribution of arterial blood and the way in which such disturbances produce their symptoms. Simple tests for vascular function and capacity in man have been devised, in order that early changes in the flow of arterial blood in the extremities can be recognized and their seriousness properly evaluated. Thus a better understanding of the pathologic-physiology of the circulation under these conditions has led to the development of more heroic methods of treatment, but with re-emphasis upon the older methods of *general care* which always have played an important part in preventing some of the more serious complications of arterial insufficiency.[9]

Active management of these disturbances should, therefore, consist of the constant and efficient utilization of all the general measures which experience has shown to be of definite value in promoting a more efficient exchange of blood in extremities, as well as those which aid in preventing infection from gaining entrance into the poorly nourished tissues of such an extremity. All too frequently, the neglect of such details of care actually is responsible for many

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of the serious complications which follow in the wake of peripheral vascular disorders.

In clinical practice, it often is difficult to make most patients realize the importance of these many precautions and even still more difficult to have them respond in an intelligent manner. Bitter experience has taught us that the fault frequently rests with us because we have not taken time, nor have we had the patience, to explain in detail the basic reasons for such an apparently complicated regimen.

Most patients readily understand that a reduction in the arterial circulation means definite impairment of the nutrition to the tissues, yet they fail to see that the many duties we have outlined to them—with respect to the position and exercise of the extremities, the avoidance of cold, the care of the skin, and the general protection from all forms of trauma—are, in reality, efforts to keep the nourishment of the tissues of the extremities at a maximum level and thus enable those tissues to combat infection in a more normal manner.

PERIPHERAL ARTERIAL DISORDERS

We have come to consider that all deficiencies of the peripheral *arterial* circulation result from a combination of vaso-occlusion and vasospasm, and that the clinical characteristics of any vascular disorder will depend upon the relative part which each of these factors plays in the production of the arterial insufficiency.

In general, degenerative arterial processes of major arteries (arteriosclerosis obliterans) present relatively little vasospasm in the arterial bed; while acute arterial accidents and sympathetic nervous system disturbances (Raynaud's syndrome) produce high grade peripheral vasospasm. The chart (figure 1) illustrates the degrees of vasospasm and vaso-occlusion which usually characterize the common clinical forms of peripheral vascular disorders. From the therapeutic standpoint, all vascular disorders which are predominently due to peripheral vasospasm are greatly benefited by the chemical or surgical interruption of the sympathetic pathways to the part; while those vascular disorders which are due largely to peripheral vaso-occlusion, without significant vasospasm,

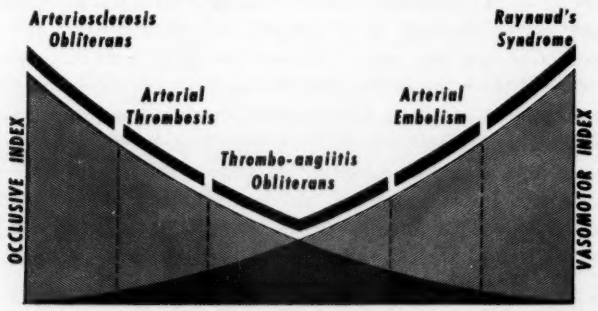

FIG. 1. Drawing showing the relative degrees of vaso-occlusion and vasospasm usually observed in the various clinical forms of arterial insufficiency in human extremities.

are only permanently benefited by enhancement of the collateral arterial circulation of that extremity.

Patients who continue to use tobacco in any form usually receive little benefit from any of the presently available vasodilating drugs. It is much more difficult to produce vasodilatation by chemical means in the normal *lower extremity* than in the normal *upper extremity*, and most difficult to bring about satisfactory degrees of vasodilatation in the extremity with obliterative arterial disease. This explains why most of the vasodilating drugs—such as papaverine, pancreatic extracts, calcium gluconate, thiamine hydrochloride, dioxyline phosphate, nicotinic acid, roniacol tartrate, aminophylline, sodium salicylate, hypertonic saline solution, and ACTH—are not effective in the treatment of patients with advanced obliterative arterial disease of the lower extremities.[3]

Spinal anesthesia and novocainization of the regional sympathetic ganglia give temporary relief of peripheral vasospasm and set the stage for the development of a collateral arterial circulation. Lumbar sympathetic ganglionectomy is the most effective means available for the production of a long-acting peripheral vasodilatation to a specific part. This vasodilatation selectively reduces the resistance to the flow of blood in the area of the body in which an increased blood flow is most needed. At the present time, it cannot be said whether or not the operation affects the collateral arteries directly, but there is clinical evidence that the nutrition of the tissues improves significantly with the passage of time following sympathectomy. Thus, it is most probable that in some way it favors the development of the collateral arterial circulation.

The restoration of the function of an injured or diseased major artery is of prime importance. Acute occlusion of a large peripheral artery by embolism, therefore, should be considered as a surgical emergency and no time should be lost in preparing the patient for the surgical removal of the obstruction within the artery. In seriously ill patients, the procedure should be done under local anesthesia and, with modern vascular surgical methods, even an embolus at the bifurcation of the aorta can be removed successfully through arteriotomy wounds in both common femoral arteries at the groin (figure 2).

Injuries to large blood vessels are not common, but there is a high incidence of complications[4] which usually can be avoided by adhering to a few basic principles. These principles of definitive treatment of fresh injuries of large blood vessels are: immediate control of hemorrhage by measures that are in themselves innocuous, treatment of shock without aiming to restore the level of blood pressure to normal before operation, early surgical exposure of the injured vessels, discrimination between vessels that may be ligated by preference and those in which restoration of continuity is obligatory, proximal and distal control of bleeding from the injured artery, avoidance of tension on the suture line or constriction of the artery, and avoidance of postoperative hypotension.

Traumatic arterial spasm most probably is due to direct injury to an artery and cannot be relieved until the source of irritation is removed. This spasm is not mediated through the autonomic nervous system but is a function of a constricting mechanism inherent in the artery. Treatment, therefore, must be

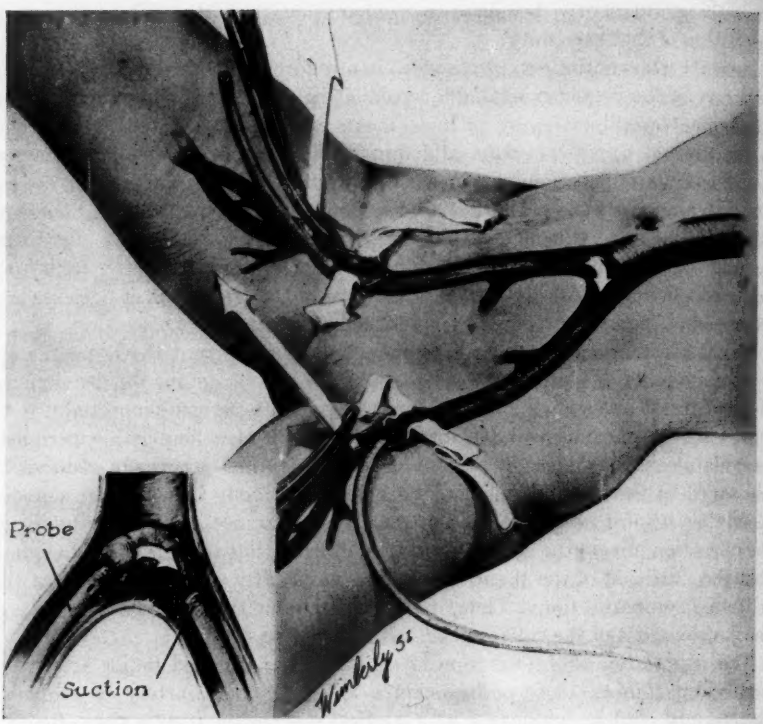

FIG. 2. Pathologic physiology of arterial insufficiency in extremities. Method of dislodging an embolus at the bifurcation of the aorta and the removal through one of the common femoral arteries in seriously ill patients.

based on direct surgical exposure of the artery in search of the offending agent. Bathing the artery in a 2 per cent solution of papaverine is of value in relaxing the artery.

The re-establishment of the lumen of large arteries which show segmental arteriosclerosis has been heralded by a few American surgeons since dos Santos,[10] Bazy,[1] and others first introduced the procedure in Europe. We have not been very enthusiastic about the procedure of thrombo-endarteriectomy since we find most patients have more widespread arteriosclerosis which would not be benefited by such local procedures. Arteriectomy, as suggested by Leriche,[8] is of value in the management of occlusive arterial disease of the popliteal artery in elderly patients.

We must bear in mind that direct injury to large arteries may result from compression against the shaft of the bone, puncture by spicules of the fractured bones, and stretching from sudden dislocation of joints or soft tissues. The faulty placement of metallic pins or screws to hold fragments of long bones in

place also takes a toll of limbs because of secondary, and many times unsuspected, injury to some large artery. Aneurysms of various types are occasional complications of skeletel injuries.

When large arterics are encased in dense fibrous sheaths, such as in the popliteal space, the sudden dislocation of the knee joint may tear the popliteal artery. Any accumulation of blood in the popliteal space warrants immediate surgical exploration of the area and the decompression of the space. If the artery is damaged, an attempt should be made to repair it; but if the vein is injured, it *always* should be ligated. Serious, and sometimes fatal, pulmonary embolism may follow an attempt to repair a large vein.

Pulsating hematomas occur when the major artery has been lacerated. When a large artery has been completely severed, considerable internal hemorrhage usually results; but contraction of the cut ends of the artery favors thrombosis and complete occlusion, and aneurysms usually do not form. Tangential injuries to large arteries always give rise to pulsating hematomas, which later develop into simple arterial aneurysms. If a spicule of bone, or any foreign object, should penetrate both the artery and the vein, a pulsating hematoma may develop at first, but this rapidly assumes the clinical characteristics of an arteriovenous aneurysm.

The treatment of simple aneurysms depends upon the location and the kind of arterial aneurysm which has developed as a result of the vascular injury. The saccular aneurysms usually can be excised and the major artery reconstructed by lateral or end to end suture technics. Fusiform aneurysms usually require excision with ligation of the proximal and distal ends or the replacement of the damaged segment of the artery by a vascular graft.[2] Our method of obliterating arteriosclerotic aneurysms in elderly patients has been by extrasaccular ligation of the incoming and outgoing arteries, with intrasaccular suturing of the genicular arteries. We have not found it necessary to do a lumbar sympathetic ganglionectomy before or after the obliteration of popliteal aneurysms in a large series of elderly patients.[5]

Simple aneurysms and pulsating hematomas which do not cause ischemia of the extremity should be treated conservatively for many weeks, but those which cause marked ischemia in the distal parts of the affected extremity must be treated immediately by surgical exploration of the area and repair of the injured artery, whenever that is possible. Delay will only give rise to irreparable changes in the soft tissues of the extremity, due to the ischemia.

Arteriovenous aneurysms should be treated conservatively for several weeks before definitive surgical treatment is undertaken, unless the strain on the heart is so great that danger of cardiac decompensation exists. Ligation of the major vein proximal to the aneurysm may relieve the load on the right heart temporarily and give time for the development of collateral arterial circulation. The reconstruction of the artery and the obliteration of the vein has been found superior to the quadruple ligation and excision of the arteriovenous aneurysm, as we formerly advocated.

PERIPHERAL VENOUS DISORDERS

For many decades venous thrombosis has been recognized as a common, and often serious, complication of chronic heart disease, debilitating illnesses, trauma, childbirth, and surgical operations. Recent surveys have shown a relative increase in the incidence of venous thrombosis, in spite of the widespread interest in the problem and the many therapeutic measures which have been used to combat it or prevent its serious sequelae.

Venous thrombosis results from a variety of mechanisms which may operate at the same time and interdependently.[6] There is no way of accurately determining in advance when or where venous thrombosis will take place in a given patient; consequently, many of our therapeutic measures are instituted only after the process has become established. Anticoagulants are of importance as prophylactic agents against venous thrombosis, and they also have proved to be valuable in limiting the process of thrombosis. The use of anticoagulants, however, is not without danger and their use must be governed by regular and accurate laboratory studies upon the clotting mechanisms of the blood.

As far as we[7] can determine, no work has demonstrated conclusively that any form of bacteria, fungus, or virus is responsible for the inflammatory reaction in any type of venous thrombosis, other than the suppurative types of thrombophlebitis. It is true that the clinical picture often is typical of an infectious process, with swelling, redness, local heat, tenderness, pain, malaise, fever, tachycardia, and increased sedimentation rate with elevation of the white cell count of the blood. Most clinicians have given large amounts of antibiotic substances in the management of thrombophlebitis solely because of this classic clinical picture. In our experience, antibiotic substances rarely have been needed.

The complications resulting from *superficial* venous thrombosis, however, usually are not serious. Nevertheless, the constant pain from the areas of thrombosis, the long period of disability, and the cost of prolonged conservative treatment should justify a reconsideration of our present conservative therapy of this clinical entity. Pulmonary embolism is rare when the venous thrombosis is limited to the saphenous varices or veins; nevertheless, it is possible for the thrombosis to extend proximally and enter the femoral vein at the saphenofemoral junction, at the saphenopopliteal junction, or through incompetent perforating veins. This may result in partial or complete occlusion of the deep veins of the extremity and, under proper conditions, even may cause pulmonary embolism.

Our experience with this type of *superficial* venous thrombosis has convinced us that the process rarely is infectious in nature; so the real danger lies in the spread of the process of thrombosis to the deep veins of the affected extremity. Anticoagulants do not alter the thrombosis which has already taken place; so if they are to be effective in limiting the spread of the thrombosis, they must be continued over a period of many weeks. The expense, as well as the difficulty of keeping the coagulability of the blood at the therapeutic level, is of considerable importance to the patient.

The complete *excision* of the involved vein with its blood clots (phlebectomy),

if done in the early stages of the process, is a simple, safe, and logical surgical procedure. If the operation is delayed until organization of the blood clots and cellular reaction about the veins becomes widespread, then the complete extirpation of the veins and the blood clots becomes much more difficult. When there still is perivenous edema, the veins can be *"shelled out"* with minimum effort and trauma to the surrounding tissues. This procedure of *superficial thrombophlebectomy* should be employed more widely, for it reduces the disability to a few days, relieves the pain promptly, reduces the total cost of the illness, and eliminates the more serious complications which occasionally follow superficial venous thrombosis in the extremities.

We believe that extension of the thrombosis or the fragmentation of the thrombus, with resulting pulmonary embolism, is related to alterations in the coagulability of the blood, as well as to the type of reaction in the wall of the vein where the blood clot exists. If the thrombus becomes adherent to the wall of the vein for only a few hours, permanent damage to the intima of the vein results. Small bits may break loose from the extending tail of the red thrombus when it builds up to the next proximal tributary where the blood stream exerts suction and torsion on the fragile clot, and pulmonary embolism takes place.

The disabling late effects of *deep* venous thrombosis, such as chronic edema, indolent ulcers, chronic eczema, pigmentation, and induration of the skin of the legs, also are responsible for serious economic loss to patients. Much has been written about these complications of *deep* venous thrombosis, and many medical and surgical measures have been used with varying degrees of clinical improvement. No single method has proved uniformly successful in all patients.

It must be emphasized that the present status of surgical therapy of *deep* venous thrombosis still is confused and leaves much to be desired before it can be considered as the ideal prophylaxis against pulmonary embolism. Even when the ligation of large veins of the extremities is promptly done, occasions will always arise when heparin and dicumarol may be valuable adjuvants in the proper management of the extending venous thrombosis.

Those interested primarily in the surgical treatment by ligation of the femoral veins do not all agree on the absolute indications for such operations. Many surgeons now are of the opinion that if a patient shows signs of pulmonary infarction, the source must be investigated carefully and, if it is found that the veins in the calf muscles of the leg are involved, the femoral vein below the profunda femoris tributary should be interrupted at once. They also believe that patients who present, for less than one week, recognizable thrombosis in the calf muscles, and who must be kept in bed for some other reason, should be treated by ligation of the superficial femoral veins. If the thrombosis is below the knee and the patient has been ambulatory for more than a week and there has been no extension of the thrombosis into the thigh, then the superficial femoral vein should *not* be ligated.

Ligation of the femoral vein below the profunda femoris tributary when the profunda femoris is patent results in relatively little edema; while ligation of the femoral vein above the profunda femoris tributary usually results in considerable

edema. We also believe that ligation of the femoral vein following aspiration may give rise to thrombosis of the previously unobstructed collaterals and thus produce massive edema.

While edema, cyanosis and pain may persist, or even increase, after iliac or femoral vein ligation, it is impossible to determine how persistent these signs and symptoms might have been had no ligation been done.

Ligation of the femoral vein below the profunda femoris branch occasionally is indicated after pulmonary infarction has taken place and the site of the thrombus definitely is localized in the veins of the calf muscles, the thigh is not swollen, and the femoral vein is not tender. Much less of an indication for ligation of the femoral vein exists when the patient has fully developed iliofemoral thrombosis; when the femoral vein is tender, and the clot within the vein is adherent and difficult to extract even by suction.

As a life saving procedure in patients with repeated attacks of pulmonary embolism due to rapidly spreading venous thrombosis in the femoral and iliac veins, the surgeon occasionally must give serious consideration to ligation of the inferior vena cava. It is true that further emboli could arise from the proximal stump of the inferior vena cava or the ovarian or accessory lumbar venous pathways, yet many lives have been saved by this heroic procedure. The subsequent disability may be considerable, but usually only moderate edema of the legs follows the ligation of the inferior vena cava. This can be controlled satisfactorily by the use of properly fitted heavy-weight elastic stockings.

Our experiences have led us to conclude that the proper use of anticoagulants is a satisfactory prophylactic measure against pulmonary embolism when: (1) the source of the emboli is not detectable clinically, (2) venous thrombosis and pulmonary embolism have occurred before operation, and (3) chemical tests reveal hyperprothrombinemia.

SUMMARY

All the present methods of therapy for peripheral vascular disorders have their limitations and it is not yet possible to assure every patient of complete recovery, even if all methods of treatment were to be employed simultaneously.

The complications of vascular obstruction in extremities are largely responsible for the high mortality and morbidity rates of these complicated disorders of peripheral blood circulation; therefore, the early recognition of vascular dysfunction and the institution of active treatment early in the course of the disturbance will give the best chances for satisfactory recovery. We always must remember that the ultimate result of high grade ischemia is gangrene of the soft tissues, amputation and permanent disability; while the ultimate result of progressive venous thrombosis is pulmonary embolism, or renal failure, and death of the patient.

REFERENCES

1. Bazy, L.: Sur l'endartériectomie désoblitérante, Mém. Acad. de chir. *74:* 109 (Feb. 11–18) 1948.
2. Clear, J. J., and Herrmann, L. G.: Operative treatment of peripheral aneurysms, Arch. Surg. *63:* 452 (Oct.) 1951.

3. Cranley, J. J., Herrmann, L. G., and Preuninger, R. M.: Evaluation of factors which influence the circulation in extremities with obliterative arterial disease, Surgery *34:* 1076 (Dec.) 1953.
4. Herrmann, L. G., and Buchman, J. A.: Complications resulting from injuries to major arteries, Surgery *26:* 59 (July) 1949 (see also J. Indiana M. A. *43:* 21 (Jan.) 1950; and Arch. Surg. *67:* 153 (Aug.) 1953).
5. Herrmann, L. G.: Surgical treatment of arteriosclerotic popliteal aneurysms in aged patients, J. Internat. de chir. (in press) 1953.
6. Herrmann, L. G.: Peripheral Venous Thrombosis, Therapeutics in Internal Medicine, New York, Hoeber Harper, Inc., 1953.
7. Herrmann, L. G.: Phlebectomy in treatment of acute thrombosis of saphenous varices or veins, Arch. Surg. *64:* 681 (May) 1952.
8. Leriche, R.: De l'artériectomie dans les artérites oblitérantes des vieillards d'après 144 observations, Bruxelles-méd. *26:* 471 (May 4) 1946.
9. Reid, M. R.: General care of peripheral vascular diseases, Ann. Surg. *96:* 733 (Oct.) 1932.
10. dos Santos, J. C.: Sur la désobstruction des thromboses artérielles anciennes, Mém. Acad. de chir. *73:* 409 (May 28–June 4) 1947.

SURGICAL ASPECTS OF HYPERPARATHYROIDISM

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Prior to any detailed consideration of those aspects of hyperparathyroidism that are of particular interest to the surgeon, it would appear desirable, since this condition still is being largely overlooked, to review briefly some of its more important clinical aspects.

CLINICAL ASPECTS

Hyperparathyroidism manifests itself through complications involving the skeleton, the urinary tract or both. Until some 20 years ago, for practical purposes, only the skeletal manifestations were recognized, so that hyperparathyroidism and generalized osteitis fibrosa cystica were virtually synonymous.[4] The renal complications were first stressed by Albright and associates[2], in 1934, who also established virtually all of the diagnostic criteria employed at present. By means of these criteria, which were entirely chemical, it became possible for the first time to establish the diagnosis with certainty in the complete absence of skeletal changes.

Experience in the past 20 years has borne out amply the prediction of the Boston group that complications involving the urinary tract would prove more common and of greater importance than the skeletal changes. It has been estimated, probably reliably, that 5 per cent of all cases of urinary lithiasis and probably 15 per cent of cases of recurrent lithiasis result from hyperparathyroidism. Skeletal complications sufficiently advanced to cause symptoms and to be recognizable roentgenologically, conversely, are unusual. In the series at the Mayo Clinic, less than 25 per cent of the patients had skeletal complications, whereas more than 80 per cent had complications involving the urinary tract. Regardless of the severity of hyperparathyroidism, if the intake of calcium exceeds its loss, demineralization of bones does not occur[1]. The skeletal changes, which range from none through varying degrees of demineralization to advanced generalized osteitis fibrosa, depend entirely on the magnitude and duration of the negative balance of calcium.

In contrast, at least until the development of renal insufficiency, a continuously increased excretion of both calcium and phosphate in the urine occurs in all cases. Stones may and do form at any time and recurrence of stones is to be expected. Calcium may be deposited also in the renal tubules, a condition known as *"renal calcinosis."* It is less common, fortunately, than lithiasis, since it is associated invariably with impairment of renal function. Impairment of renal function, with or without roentgenographically evident calcinosis, is the most serious complication of hyperparathyroidism. Beyond a certain stage,

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which as yet cannot be defined with confidence, the impairment of renal function is progressive in spite of cure of the hyperparathyroidism[5]. Death results ultimately from hypertension or from renal insufficiency. If the patient is to be cured, the hyperparathyroidism must be recognized and controlled before renal function is impaired to the degree that it becomes progressive.

The possibility of hyperparathyroidism should be seriously considered in all cases of urinary lithiasis in which the stones contain calcium, in all cases of renal calcinosis, in cases of polyuria and polydipsia and in all cases of generalized demineralization of the skeleton. The symptoms and clinical findings are never sufficient to establish the diagnosis, which in all cases depends on the demonstration of characteristic metabolic changes in blood and, to a lesser extent, in urine. These include hypercalcemia, hypophosphatemia and an increased excretion of both calcium and phosphate in the urine.

The first three of these changes are of importance diagnostically. If renal function is normal, the three changes should be demonstrable. With impaired renal function, the value for phosphate may be within or greater than the physiologic range. Similarly, increased excretion of calcium in the urine may not be demonstrable in the presence of renal damage. Thus, the one constant finding in all cases of hyperparathyroidism is hypercalcemia. The upper limit of the physiologic range for calcium has been regarded for a long time at the Mayo Clinic as 10.5 mg. per 100 cc. of serum. The values for calcium in the series of cases at the clinic ranged upward from this value. In almost 10 per cent of cases the value for calcium was less than 11 mg.; in more than 40 per cent of cases, it was less than 12 mg. It is obvious that determinations of calcium must be extremely accurate if disease of minimal severity is to be recognized. The demonstration of hypophosphatemia and hypercalcuria aids materially in confirming the diagnosis, particularly in cases of less severe disease without evidence of impaired renal function.

The most important diagnostic problem in the field is the differentiation of renal lithiasis due to hyperparathyroidism from renal lithiasis of other origin. In all cases of hypercalcemia the second problem of differentiating hyperparathyroidism from all other hypercalcemic states arises. The difficulties may be extremely great and much expertness is required before the diagnosis can be made with confidence.

Further discussion of the clinical aspects is beyond the scope of the present paper. They have been admirably covered by Albright and associates, by Keating and Cook[10] and by Albright and Reifenstein[3]. In every institution where case finding has been particularly successful, there has been an unusual interest in the metabolism of calcium in general and in hyperparathyroidism in particular. Unless there is a joint interest in the condition on the part of internist, urologist and clinical pathologist, the condition will be recognized rarely and the diagnosis practically never made with confidence.

SURGICAL ASPECTS

The treatment of hyperparathyroidism is surgical removal of the hyperfunctioning parathyroid tissue. It is made difficult by the minute size and

varied locations of the parathyroid glands and by variations in the pathologic lesions. A comprehensive knowledge of the surgical pathology and surgical anatomy of the parathyroids is essential if treatment is to be successful[5].

Surgical Pathology. Hyperparathyroidism may result from a single adenoma, from multiple adenomas, from primary or wasserhelle hyperplasia or from carcinoma[13]. The commonest lesion by far, accounting for approximately 80 per cent of cases, is the single adenoma. Adenomas vary in size from that of a normal parathyroid gland to relatively large tumors weighing more than 100 Gm. In the series at the Clinic the weight of the adenoma was less than 100 mg. in 7 per cent of cases, less than 500 mg. in 35 per cent of cases and less than 1 Gm. in more than 50 per cent of cases. Adenomas that weighed more than 10 Gm. were found in less than 10 per cent of cases. The tumors are recognizable grossly by their characteristic yellowish-brown color, which is different from that of lymph nodes, thymus or thyroid. Multiple adenomas may be expected in somewhat less than 10 per cent of cases; they do not differ grossly or microscopically from single adenomas. Two, three or even all four of the parathyroids may be involved. When more than two glands are involved, the patients usually have a curious and unusual polyendocrine syndrome in which there are associated tumors of the pituitary and hyperfunctioning islet cell tumors of the pancreas[11].

Microscopically, the most obvious difference between adenomatous and normal parathyroid tissue is the almost complete absence of stromal fat within adenomas. The parenchymal cells differ little from those of the normal gland, and chief, wasserhelle and oxyphil cells, along with transitional forms, are present in adenomatous tissue. One type of cell often predominates, permitting histologic classification; however, this has little or no significance from a clinical standpoint.

Primary, or wasserhelle, hyperplasia occurs in another 10 per cent of cases. All parathyroid tissue is involved. The hypertrophied cells, which are monotonously similar, have a clear featureless cytoplasm; this gives rise to the term "*wasserhelle*," or "*water-clear*," cell hyperplasia. By contrast, the nucleus, which stains densely, is prominent. The weight of hyperplastic tissue in the cases at the clinic has varied from 760 mg. to 52.5 Gm. Hypertrophy and hyperplasia of this degree are seen in no other condition. An increase in size of a thyroid weighing 20 Gm. proportionate to that in the case in which 52.5 Gm. of parathyroid tissue was removed would produce a goiter weighing some 8,000 Gm.

The remaining lesion that can cause hyperparathyroidism is true carcinoma, capable not only of recurring locally but of metastasizing. Fortunately, carcinomas are rare, since in the great majority of cases so far reported, the lesions have proved incurable. Carcinomas may be indistinguishable microscopically from benign adenomas[13]. Benign lesions have no tendency to infiltrate beyond their capsules, whereas this has been a common feature of carcinomas.

Variations in the pathologic lesions have most important surgical connotations. Adenomas less than 500 mg. in weight may be extremely difficult to find, not only because of their small size but also because they tend to be caught between neighboring anatomic structures or within sulci of the thyroid. Because of the possibility of multiple adenomas, the surgical procedure should not be terminated

when one adenoma is found. Primary hyperplasia is usually easily recognizable grossly since, as a rule, all four glands are greatly hypertrophied. In an occasional case, however, in spite of the fact that all parathyroid tissue is involved, the different glands may vary tremendously in size. Thus, association of a single enlarged gland with three relatively small glands, or any other combination, is possible. Primary hyperplasia must be recognized at the time of operation since the hyperplastic tissue must be resected subtotally, preserving from 30 to 200 mg. of vascularized tissue. If only one gland is removed or if a less nearly complete resection is carried out, hyperparathyroidism will persist.

Surgical Anatomy. The only surgical approach adequate to meet the contingencies imposed by the different lesions is the methodical demonstration of the four parathyroid glands. Rarely fewer than four glands may be present because of fusion of the two glands on one side. In this case the fused gland is approximately doubled in size. Also, more than four glands may be encountered. The supernumerary gland or glands are in the immediate vicinity of one of the normal glands. The possibility of an adenoma in a widely aberrant supernumerary gland need not be feared greatly.

The recognition of normal parathyroids, regardless of many statements to the contrary, is not particularly difficult. The glands, although tiny, are definite structures with a delicate capsule, beneath which fine vessels spread from the hilus. They are usually flattened, elongated or otherwise distorted by neighboring structures. They are recognizable, as are adenomas, by their characteristic yellow-brown color, which, together with their location, size, form and structure, makes gross identification reasonably certain. In hyperparathyroidism, uninvolved glands are more or less atrophic. Atrophied glands may not be materially smaller than normal glands but it is far more difficult to recognize them because they tend to lose the characteristic color of parathyroid tissue. With experience, identification of atrophic glands is still usually possible without biopsy.

A final difficulty in the finding of parathyroid glands has to do with variations in location, chiefly as a result of their embryologic migration[8]. The parathyroids develop from the third and fourth pharyngeal pouches and descend with the thyroid (superior pair) and thymus (inferior pair). The descent of a superior gland may be arrested at any level from above the larynx to the inferior pole of the thyroid. The descent of an inferior gland begins rostrad to that of the superior gland and may continue to the level of the arch of the aorta. The four glands are always within the visceral compartment of the cervical fascia or its inferior prolongation in the mediastinum. In addition to displacements due to faulty or arrested embryologic migration, an enlarged gland may be displaced toward or into the mediastinum by the same forces that cause a goiter to sink into the thorax[12]. In such cases a vascular pedicle connects the displaced parathyroid with the arterial system of the thyroid. The pedicle, which may be surprisingly large, usually branches from the thyroidal vessel close to the thyroid. Vascular pedicles are of tremendous surgical importance since, by following them, displaced adenomas may be found and removed under direct vision without resorting to mediastinotomy.

While a gland may be located at any level from above the larynx to the pericardium, widely aberrant glands are not common. Gilmour[9], in a study at necropsy of the location of the parathyroid glands in 428 cases, found that more than 90 per cent of superior glands were located on the posterior or medial surface of the thyroid above the lower third of the lobe. Approximately 95 per cent of inferior glands were located within the immediate vicinity of the lower poles of the thyroid and another 3 per cent were between 3 and 6 cm. below the lower poles. The findings among surgical cases are not dissimilar. In the series of approximately 180 proved cases at the Clinic, formal dissection of the anterior superior mediastinum was necessary in 2 cases only. In 2 other cases, adenomas well below the level of the cervical dissection were removed safely by gentle traction on the vascular pedicle. The possibility of an intrathyroidal parathyroid adenoma should not be forgotten. There have been 2 such cases in the series at the clinic.

It may be concluded that the vast majority of parathyroid glands are located on or near the thyroid. Similarly, most adenomas are in the cervical region or uppermost part of the mediastinum, where they can be found and removed under direct vision through the cervical incision. Before concluding that mediastinal exploration is necessary, the surgeon should realize that the chances are probably 50 to 1 that the tumor is not within the mediastinum but has been overlooked during cervical dissection.

Surgical Procedures. The cervical region is explored through an incision for thyroidectomy. Below the strap muscles the field must be kept bloodless at all cost, since the color by which parathyroid tissue is recognized is lost completely in blood-stained tissues[7]. After the lobe of the thyroid on the first side has been widely mobilized, the areolar tissue is carefully separated from the capsule of the thyroid and from the superior and inferior thyroid arteries. The inferior thyroid veins are stripped clean and the recurrent laryngeal nerve is exposed throughout the operative field. A specific search then is made for each of the two parathyroids on the side. Gross identification is sufficient at this stage of the procedure. After a test is made of the function of the recurrent laryngeal nerve, a similar dissection is carried out on the opposite side. During the course of the dissection, any pedicles leading into the mediastinum will have been found and followed to the adenoma if this can be done under direct vision. In all cases, bilateral dissection should be completed.

Adenomas are removed totally. In cases of primary hyperplasia, the hyperplastic tissue is resected subtotally, leaving a vascularized fragment of tissue perhaps three times (estimated weight = 100 mg.) the size of a normal parathyroid to prevent tetany[6]. In theory, carcinomas should be removed as radically as possible, along with the regional lymph nodes. As previously mentioned, success in treating parathyroid carcinomas has been most limited. In most of the reported cases the lesions were not recognized at the original procedure as carcinomatous, and the original resection was not radical. It is probable that a more aggressive surgical attack at the initial operation well may be followed by improved results in the future.

If no lesion has been found after complete bilateral dissection, absolute identification of the parathyroids by biopsy, after provisional gross identification, is necessary to prove which of the glands is missing. Great care must be taken to avoid destruction of the gland during biopsy. The missing gland is then sought by extension of the dissection to include the entire region in which the gland theoretically may be situated. The most likely location of a concealed superior gland is medial to the thyroid or between the trachea and the esophagus. That of a missing inferior gland is caudal to the inferior pole, closely associated with one of the inferior thyroid veins, or the cervical extension of the thymus or closely applied to the trachea. It is, of course, often impossible to find three atrophic parathyroids and to prove that the fourth is missing. Nevertheless, the number of glands found during the course of a fruitless cervical dissection is a measure of the completeness of the dissection. The fewer the glands identified, the less the certainty that the adenoma is within the mediastinum and the greater the probability that it has been missed during the cervical dissection.

Mediastinal Dissection. Formal dissection of the anterior superior mediastinum is never undertaken until several months after the cervical dissection[8]. It is wise to re-establish the diagnosis before proceeding with mediastinotomy, since a small lesion may have been removed or destroyed unknowingly during the course of the cervical dissection. The mediastinum is exposed by splitting the sternum, as for resection of the thymus. The dissection, as in the neck, must be bloodless and meticulous. If the adenoma cannot be found, the thymus should be removed, since an inferior parathyroid may be located within the thymus.

Postoperative Course and Complications. The only technical complication that requires mention is injury to the recurrent laryngeal nerves. As already indicated, both nerves should be exposed routinely throughout their course in the operative field. Injury to one nerve usually results in no impairment of the airway. The combination of one fixed vocal cord and tetany may result in stridor, which should respond promptly to the intravenous injection of calcium lactate. As with stridor from any cause, if an adequate airway is not secured immediately, tracheotomy should be carried out without delay.

Transient tetany is rather common. Symptoms of tetany may result from abrupt decrease in the level of calcium from the hypercalcemic to the physiologic range. In other cases, the values for calcium may decline temporarily to the range encountered in tetany. In either case the tetany is mild and transient, and usually requires little treatment. If all parathyroid tissue is destroyed, the tetany obviously becomes permanent and treatment is necessary. Parathyroid preparations (such as parathormone) practically never are used, and intravenous injections of calcium lactate or gluconate are required only as emergency measures to control severe symptoms or to prevent convulsions. Values for serum calcium are restored to the normal range and maintained there by the combination of calcium lactate or gluconate by mouth and dihydrotachysterol (AT 10; hytakerol) in appropriate dosages.

An unusually severe type of tetany (bone-hunger tetany of Albright) is to be expected postoperatively in cases of advanced osseous disease. It results, pre-

sumably, from the rapid deposition of calcium and phosphorus in the demineralized bones. Extremely aggressive treatment, including the intravenous administration of calcium, is necessary for even partial control of the symptoms.

PROGNOSIS

The ultimate results after treatment depend on the status of the kidneys. The demineralized skeleton slowly regains its normal strength and the internal architecture of the bones reverts toward normal, although gross deformities persist. As previously mentioned, renal function may continue to deteriorate, if impaired beyond a certain point, in spite of cure of the hyperparathyroidism. As far as is known at present, hyperparathyroidism resulting from adenomas and primary hyperplasia does not recur, although a few instances have been reported of subsequent development of a second adenoma in another gland.

SUMMARY

The possibility of hyperparathyroidism should be considered in all cases of renal lithiasis in which the stones contain calcium, in all cases of renal calcinosis, in cases of generalized demineralization of the skeleton and in cases of polyuria and polydipsia. The diagnosis is established by means of chemical tests that must be exceedingly accurate. The only effective treatment is surgical removal of the adenoma or adenomas, the hyperplastic tissue or the carcinoma producing the excessive amounts of parathyroid hormone. The surgical problems, which have to do with the recognition of normal and atrophic parathyroid glands, the minute size of many adenomas, the different pathologic lesions that may be encountered and the variation in location of the parathyroids, have been discussed.

REFERENCES

1. Albright, F.: Parathyroids—physiology and therapeutics, J. A. M. A. *117:* 527 (Aug. 16) 1941.
2. Albright, F., Aub, J. C., and Bauer, W.: Hyperparathyroidism; a common and polymorphic condition as illustrated by 17 proved cases from one clinic, J. A. M. A. *102:* 1276 (April 21) 1934.
3. Albright, F., and Reifenstein, E. C., Jr.: The Parathyroid Glands and Metabolic Bone Disease: Selected Studies, Baltimore, The Williams & Wilkins Company, 1948, p. 15, 73.
4. Barr, D. P., Bulger, H. A., and Dixon, H. H.: Hyperparathyroidism, J. A. M. A. *92:* 951 (March 23) 1929.
5. Black, B. M.: Hyperparathyroidism, Springfield, Illinois, Charles C Thomas, 1953, p. 68.
6. Black, B. M., and Sprague, R. G.: Hyperparathyroidism due to diffuse primary hypertrophy and hyperplasia of parathyroid glands; report of case, Proc. Staff Meet., Mayo Clin. *22:* 73 (Feb. 19) 1947.
7. Churchill, E. D., and Cope, O.: Parathyroid tumors associated with hyperparathyroidism; 11 cases treated by operation, Surg., Gynec. & Obst. *58:* 255 (Feb. 15) 1934.
8. Cope, O.: Surgery of hyperparathyroidism; occurrence of parathyroids in anterior mediastinum and division of operation into 2 stages, Ann. Surg. *114:* 706 (Oct.) 1941.
9. Gilmour, J. R.: Gross anatomy of parathyroid glands, J. Path. & Bact. *46:* 133 (Jan.) 1938.
10. Keating, F. R., Jr., and Cook, E. N.: Recognition of primary hyperparathyroidism; analysis of 24 cases, J. A. M. A. *129:* 994 (Dec. 8) 1945.
11. Underdahl, L. O., Woolner, L. B., and Black, B. M.: Multiple endocrine adenomas; report of 8 cases in which parathyroids, pituitary and pancreatic islets were involved, J. Clin. Endocrinol. & Metab. *13:* 20 (Jan.) 1953.
12. Walton, A. J.: Surgical treatment of parathyroid tumours, Brit. J. Surg. *19:* 285 (Oct.) 1931.
13. Woolner, L. B., Keating, F. R., Jr., and Black, B. M.: Tumors and hyperplasia of parathyroid glands; review of pathological findings in 140 cases of primary hyperparathyroidism, Cancer *5:* 1069 (Nov.) 1952.

ACUTE OBSTRUCTION OF THE SMALL BOWEL

THE EXPERIENCE OF A SMALL GENERAL HOSPITAL WITH FIFTY RECENT CASES

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The 50 cases of acute obstruction of the small bowel which form the basis of this communication make a small series when they are compared with the hundreds of cases which are included in many of the other reported series of this condition. There are, however, a number of reasons for analyzing them, aside from the overwhelming, if entirely personal, reason that a surgeon should pause at intervals to take stock of the work being done by himself and his immediate associates.

These 50 cases represent the work of a single surgical service, consisting of a senior surgeon, his assistant, and the surgical residents working under their supervision. They also—which is more important—represent the recent experience with this type of intestinal obstruction in a small, nonuniversity hospital, located in a small community, but operating, so far as possible, on the general principles which would be observed in university hospitals in large centers of population. In this country the majority of operations are not done in university-connected hospitals in large medical centers. It is impractical that they should be. The size of a hospital, however, should have nothing to do with the quality of the work done in it. Perhaps certain concessions must be made to expediency in small, nonteaching hospitals, but it is never necessary, nor is it ever permissible, to compromise with fundamental surgical principles.

There were errors made in the management of these 50 cases, as will be pointed out, but it is fair to say that the errors had nothing to do with the fact that the cases were managed in a small, nonteaching hospital. There were only two deaths, and a 4 per cent mortality rate, which is also fair to say, is creditable for a condition as potentially lethal as acute obstruction of the small bowel. There were no deaths in the 7 patients who were treated by the surgical residents. This confirms what many other reports on all types of cases have proved—that it is perfectly safe to permit properly trained residents to assume the responsibility for major surgical diseases.

BASIC DATA

No instances of paralytic ileus are included in these 50 cases. This condition, we believe, is not surgical and in our hands it always is treated by intestinal decompression and adjunct supportive measures.

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The series also does not include any instance of small bowel obstruction not confirmed by surgical exploration. Over the 7 year period (1947 to 1953) covered by this analysis an even larger number of patients with this condition were treated conservatively and escaped surgery, at least for these particular episodes. Whether some of them will return in the future with recurrent obstruction only time will tell. This is a condition which tends to recur. Three of the 50 patients had such histories and 1 of them had been hospitalized three times before surgery was eventually necessary. Surgery, of course, does not always solve the problem permanently. One of the 48 survivors in this series returned in three months with a second obstruction, which responded to conservative therapy. Another, after remaining well for five years, returned with a second obstruction associated with two perforations of the bowel, directly attributable to delay in seeking medical aid. She recovered after lysis of adhesions and closure of the perforations.

With the exceptions just noted, these 50 cases represent almost every variety of small bowel obstruction: complete (in 49 instances) and partial; primary and recurrent acute; rapidly and slowly progressive; of varied etiologic origin, including adhesions following operation and of unknown origin, hernias of all types, volvulus, intussusception and neoplasm. The series also represents all gradations of physiologic derangement.

Etiologic Factors. Etiologically these 50 cases are divided as follows:

Twenty one cases of adhesive obstruction, 17 of which followed immediate or remote surgery and 8 of which required resection.

Twenty cases of external hernia (15 inguinal, 2 femoral, 2 ventral and 1 umbilical), 2 of which (both inguinal) required resection.

Three cases of internal hernia, including one hernia of the diaphragm, 1 of which required resection.

Two cases of intussusception, with 1 resection.

Two cases of volvulus, with 1 resection.

One case of mesenteric vascular occlusion, with resection.

One case of adenocarcinoma of the jejunum, with resection.

The wide variety of conditions included in these 50 cases make statistical computations of little value. Clinical analysis, however, is of considerable value, because the varied etiology indicates that the series, represents a true cross section of small bowel obstruction as it is encountered in general practice. The distribution of etiologic factors is, in fact, about what it would be in larger series.

In 1937, in a comparative analysis of three series of intestinal obstructions totaling 715 cases, Boyce[3] called attention to the increased incidence, in the most recent series, of obstructions caused by postoperative adhesions and remarked that every abdominal operation provided a potential future obstruction. Hernia formerly was the chief cause of small bowel obstruction, but in the last 10 to 15 years that has ceased to be true, obviously because of the increasing incidence of intra-abdominal surgery. The implication of this change in etiologic factors is not always stressed as it should be. It is the responsibility which it places upon all surgeons to refrain from surgery which is not essential, and to carry out all

intra-abdominal surgery by an atraumatic technic and with strict attention to all technical details.

Sex, Race, and Age. Like all series of small bowel obstructions, this series includes both males and females (28) the largest group of males (15) presenting inguinal hernias and the largest group of females (15) presenting adhesive obstruction, most often after pelvic surgery. The series differs, however, from many recorded series in that it includes negroes. The 21 patients of this race presented various types of obstruction and accounted for 11 of the 15 obstructions caused by inguinal hernia. Over the period covered by this study the ratio of Negro to white hospital admissions for all causes was 1:4, but the series is too small to make the larger proportion of negroes in it of any significance.

The age range was wide, from 4 months in a child with intussusception to 81 years in a Negro woman who developed adhesive obstruction 40 years after hysterectomy. During the interim she had had no trouble of any kind and recovered uneventfully after simple release of adhesions involving the ileum. Because of a four day delay before admission she was a poor risk and preparation consumed 24 hours. Other patients, incidentally, also proved that the risk of postoperative adhesions is a continuing one. One man developed his obstruction four weeks after an operation for rupture of the appendix but another developed his 13 years later. A 47 year old Negro woman had no difficulty for 20 years after a hysterectomy but became obstructed four months after radium had been applied for carcinoma of the cervical stump. Whether the adhesions were caused by the *long ago* surgery or were a reaction to the recent irradiation is not possible to say.

Six patients in the series were children under 3 years of age. One presented intussusception and the others some variety of hernia. At the other extreme, 15 patients were 60 years of age or older. Only 16 patients, including the 6 children just mentioned, were under 40 years of age. Although the series was rather heavily weighted with older persons, only one of the two deaths occurred in the upper age group, which is ordinarily supposed to carry an additional risk from the standpoint of age alone.

PRINCIPLES OF MANAGEMENT

Surgical Measures. Small bowel obstruction now is a well understood disease. Our knowledge, in fact, is such that the patient with a partial or complete obstruction, if he is operated upon promptly and if he receives the correct adjunct treatment, should recover, and usually should recover without complications. Our general plan of procedure is to operate without delay upon any patient who presents a clear cut acute obstruction of the small bowel which is thought to be of mechanical origin. When the diagnosis is doubtful, exploration is done with equally little delay. Unfortunately, as will be pointed out shortly, the diagnosis of acute small bowel obstruction is not always as simple as this statement of principles might seem to suggest.

Patients with obstructions caused by inflammatory processes arising on

the basis of a recent peritonitis or initiated by recent surgery do not always lend themselves to clear-cut decisions. Generally speaking, particularly when the obstruction follows recent surgery, conservative therapy is more desirable than a second major surgical procedure. On the other hand, the recognition of an obstruction which can be safely treated conservatively, as distinguished from an obstruction which requires surgery, often is extremely difficult, even when the patient is under constant observation.

This problem was encountered in 2 patients in this series, and 1 of them died. The first patient, a 39 year old Negro woman, had a huge fibroid of the uterus, for which hysterectomy was done, and a very large ventral hernia, in the repair of which tantalum mesh was used. The wound became infected and complete disruption occurred a month after the first operation. When secondary closure was undertaken, 4 feet of small bowel were found gangrenous and required resection. The tantalum mesh, to which the bowel was adherent, was removed with difficulty, and in the course of the manipulations, the large bowel was damaged in three areas. Repair of all injuries was made without difficulty, but transverse colostomy was done as a precaution. This is not a case upon which we look with any pride. The symptoms of intestinal obstruction were overshadowed by the more obvious symptoms arising from wound infection and disruption. The diagnostic error was compounded by the technical error at operation, by which the large bowel was injured. Although the patient was in poor condition at the conclusion of the operation, her recovery was entirely uneventful and the colostomy was easily closed two months after the second operation.

The second patient, a 12 year old white boy, had undergone appendectomy for a ruptured appendix 10 days before he required surgery for an obstruction of the ileum caused by adhesions arising from acute purulent peritonitis. The adhesions were divided, ileostomy and jejunostomy were done, and the cavity was drained. Improvement was never sustained and he died a month later of acute diffuse peritonitis and intra-abdominal abscess. It may be that his life would have been saved by wiser management at the second operation, but adjunct measures left nothing to be desired, and it does not seem unfair in this instance to list as the ultimate cause of death the fact that he was treated for abdominal pain for a week before admission by a combination of cathartics and a sulfonamide drug prescribed by a private physician.

Resection was required in 15 of the 50 cases of small bowel obstruction in this series. Sometimes, as will be noted later, it was necessary because of diagnostic error and ill-advised preoperative therapy, but in other instances it was required by the inherent nature of the pathologic process. The surgeon has no choice, for instance when the obstruction is caused by a malignant neoplasm, and similarly, he has no choice in mesenteric vascular occlusion. The only patient in the series with mesenteric vascular occlusion was a 67 year old white woman who was a poor risk when she was first seen after 27 hours of futile treatment outside of the hospital. It was necessary to resect almost 4 feet of ileum. Twenty-

four days later obstruction of the stoma developed at the site of the end to end anastomosis. The patient's prompt recovery after a second major surgical intervention is a further illustration of how greatly the risk of intestinal obstruction has decreased in recent years; 15 years ago her chance of surviving primary surgery for mesenteric vascular obstruction would have been small.

Our own preference is for the ordinary open type of resection and side to side anastomosis. We believe that so-called aseptic end to end anastomosis, strictly speaking, is not aseptic at all and it does not permit the surgeon to choose the size of the stoma.

Adjunct Measures. The preoperative and postoperative management of patients with small bowel obstruction is now so routine that no time need be spent in discussing it. As one looks back on the cases encountered 20 years or more ago, it is perfectly evident that a great many patients died because of lack of appreciation of the disturbances of fluid and electrolyte balance which occur in this condition. There is now small reason for any patient to lose his life from such a cause. Inadequate adjunct treatment was not a factor in either of the fatalities in this series. It is also not too much to say that almost without exception all the patients were properly prepared for surgery and were properly treated afterward.

In all but a few instances some sort of preoperative preparation was needed, the requirements varying from patient to patient according to the degree of dehydration present and the deficits and perversions revealed by the laboratory studies which are made as a matter of course in all small bowel obstructions. Our chief reliance for replacement therapy is a solution of sodium, potassium, calcium and magnesium chlorides with sodium lactate (ionosol D-CM), which is both efficient and convenient to use. Dextrose and amigen are used to supplement it as necessary. The liberal use of blood in these cases sharply distinguishes them from patients treated 15, or even 10, years ago. On the other hand, blood was administered only upon indication. We take the position that transfusion, in spite of its benefits, is not a method to be lightly used, and we withhold it when we believe that it is not required.

Antibiotic Therapy. Antibiotics have played their part in the sharp reduction in the mortality and morbidity rates of small bowel obstructions. They were used in one form or another in every case in this series and undoubtedly had much to do with the low mortality rate as well as with the generally smooth recoveries. Of the 48 surviving patients, only 3 developed complications. One had a wound infection, 1 a fecal fistula, and 1, as already noted, a secondary obstruction for which additional surgery was necessary.

The chief usefulness of antibiotic therapy is in the control of infection incident to the intestinal lesion and the surgical procedure and in the prevention, or control, of peritonitis incident to rupture of the bowel. Two ruptures of the bowel were found at operation and four others occurred as the friable obstructed loops were being manipulated. Fifteen years ago the death of 6 similar patients would have been prohibitive, but in this series only 1 patient who presented a

preoperative rupture lost her life and, as will be noted shortly, she could have been saved if she had been treated more wisely.

For the last year or two we have avoided rupturing the bowel at operation by decompressing it before it is manipulated. Rubber-shod clamps are placed on a suitable loop, a purse-string suture is placed, a long suction tip is introduced, and the contents of the bowel are suctioned off. The purse-string suture is then tied. The risk of rupture is greatly reduced by this maneuver, and the emptied bowel is much easier to deal with.

The routine of antibiotic therapy in intestinal surgery has changed from year to year as new and more potent agents have been introduced, and there would be small point in discussing the various methods used in this series except to say that they were altered as the new agents became available. As a rule we now use streptomycin and penicillin, resorting to neomycin however, when a particularly prompt effect is desired.

Intestinal Decompression. It is an ironic fact that the methods which have had most to do with lowering the mortality rate in small bowel obstruction are also the methods responsible for much of the mortality rate which continues to be associated with it. Improvement occurs under replacement therapy and operation is deferred until it is forced on the surgeon by deterioration in the patient's condition. Antibiotics are miracle drugs and it is sometimes forgotten that their miracles are exerted upon bacteria and that a mechanical intestinal obstruction still requires mechanical liberation.

Probably the most serious of all errors in the management of small bowel obstruction is the use of intestinal decompression as a tentative substitute for surgery. Almost from the time he introduced this method Wangensteen warned against its use in this manner, but there are few reported series in which the error is not evident. This series is no exception. We recognize the fallacy, but we still fall into it, on the surgical as well as on the medical service.

Intubation was used in this incorrect fashion in 12 patients in this series, for periods ranging from nine hours in a case of volvulus to six days in a case of adhesive obstruction. Six of the 12 patients eventually required resection, and it is quite possible that resection might have been avoided in 1 or 2 other patients if preoperative intubation had not been persisted in for too long a time. There are few surgeons who are not haunted, as we are, by the memory of patients who were subjected to greater risks than they should have been because of this error in judgment. In 1 patient in this series the mistake was fatal. The patient, a 75 year old white woman, was admitted to the medical service with bronchopneumonia and her abdominal symptoms and signs, which were of moderate severity, were attributed to the respiratory disease. The mistake became apparent at the end of 48 hours of constant intestinal decompression. By this time the patient had become a poor surgical risk and she died soon after resection of 130 cm. of gangrenous ileum which was perforated in one area.

If one puts a tube in the small bowel and 12 hours later finds a strangulation obstruction at operation, one may be sure that the strangulation was present

and was missed when the patient was first seen, or it developed while he was under observation. In either instance, the responsibility for it must be accepted. Whether in the case just described the strangulation was present when the patient was admitted, and was overlooked in the concentration of attention on the medical condition, is a matter of speculation. There is no doubt, however, that the unwise use of intestinal decompression played a major part in the fatality.

The most instructive portion of the very excellent study by Becker[1] of 407 patients with small bowel obstruction from Charity Hospital of Louisiana at New Orleans over the 10 year period ending in 1949 deals with errors in the use of intestinal intubation. It was employed in 369 patients. In 52 of these patients it was, as the author puts it, "*abused*," with the result that the mortality rate in this group of cases was 38.4 per cent, as compared to a mortality rate of 11.8 per cent for the whole series. The 52 deaths were distributed as follows: Forty three instances of strangulation obstruction, in which operation was done with 11 deaths, after at least 12 hours of decompression. These patients either had strangulation obstructions on their admission or developed them in the course of therapy. Three instances of obstruction were treated by intubation alone, in which strangulation was recognized only at autopsy. Six instances in which the patients were initially in good condition but exhibited marked deterioration as intubation was persisted in. The mortality rate of delayed surgery in this group was 100 per cent. The case in our series which has just been described fits well into this group.

Patients who need intubation the least generally are intubated with the least difficulty. Those who would be benefited the most by it frequently are intubated with great difficulty and with loss of time. These cases are becoming less frequent as more efficient modifications of the Miller-Abbott tube are introduced, but they are still being encountered. Our own preference is for the Cantor or the Harris tube.

It well may be that the physician's tendency to resort to intubation unwisely and for undue lengths of time arises from his recollection of the days when surgery for intestinal obstruction was highly lethal, chiefly because modern adjunct therapy did not exist. Replacement therapy was just coming into general use when Wangensteen began to popularize intestinal decompression, and another 10 years was to pass before adjunct measures were employed routinely. Antibiotic therapy was a still later development. It is small wonder that some physicians, when they rely on intestinal decompression instead of resorting to surgery, do not realize how radically the situation has changed. As a matter of fact, the risk of unnecessary exploration at the present time is considerably less than the risk of overlooking strangulation obstruction while employing decompression.

Another point to be borne in mind when intubation is employed as definitive treatment is that, although it may carry a patient successfully over an episode of incomplete obstruction without vascular damage, it must not be concluded

that it will be equally successful in subsequent episodes. Whenever intubation is used in this manner, the patient should be warned that the difficulty may recur, and that there is no guarantee that treatment by intubation will be even indicated, let alone will be successful, in a subsequent attack. The death warrant of many a patient has been signed by the surgeons failure to give, and the patients failure to heed, such counsel.

STRANGULATION OBSTRUCTION

Although the time element plays an important part in both the morbidity and the mortality rates of small bowel obstructions, the mere passage of time no longer, in itself, places the patient beyond salvage. McKittrick, in a discussion of Evans and Bigger's[6] paper presented in 1947, remarked that there should be no deaths in the first 18 to 24 hour period, regardless of the type of obstruction or the procedure necessary to correct it, but that a mortality rate of 25 per cent could be expected within the next 24 hour period. It is an interesting commentary on the improvement which has occurred in this condition since 1947 that in our own series, which covers the period 1947 to 1953, there was only one death in the 19 patients admitted after the 24 hour period of grace.

In the 14 patients who required resection after their obstructions developed outside of the hospital, the duration of symptoms before admission ranged from 5 to 72 hours and the duration of hospitalization before operation from 1 hour to 144 hours. The condition of 2 of these patients was good at operation, 8 were in fair condition, and 5 were in poor condition; the status of some of them, as has been pointed out, had deteriorated because of unwise persistence in conservative therapy. The single death in these 14 resections occurred in a patient in whom the delay before admission was 48 hours and the delay in hospital was the same length. On the other hand, the total delay in this case was less than in a number of other cases in which the patients survived. Obviously, the time element, important as it is, is no longer as important as it once was. Today the pathologic process per se probably plays the most important role, particularly the pathologic process in which strangulation is a primary component or in which, because of unwise management, it has become a secondary component.

A number of observers have set up criteria by which, at least in theory, strangulation obstruction of the small bowel can be distinguished from complete obstruction. Evans and Bigger,[6] for instance, believe that strangulation obstruction presents the following distinguishing characteristics: 1. The pain is sudden and the onset almost dramatic. 2. Vomiting is frequent and repeated. 3. The patient looks extremely ill from the onset. 4. Shock is an early occurrence. 5. The patient seeks the *position of relief*, tending to lie upon one side or the other and not supine, as he would lie in simple obstruction. 6. He complains of pain in the back, as McKittrick first pointed out. 7. Abdominal tenderness is constant and is sometimes associated with rebound tenderness and with generalized or local spasm. 8. A mass may be palpable. 9. Auscultation reveals the intestinal sounds to be less frequent and somewhat lower in tone and pitch

than in simple obstruction. 10. The leukocytosis tends to rise. 11. The pulse rate is likely to be above 100, although the authors' own experience did not bear out this observation. In 6 of their 16 patients it was below 100. There were five deaths in these 6 patients.

Evans and Bigger[6] did not consider roentgenograms of differential diagnostic value in strangulation obstruction, but Cole, in a discussion of Bollinger and Fowler's[2] paper, took the position that they are very useful indeed. He listed the following as distinctive roentgenologic observations in strangulation obstruction: 1. The pattern is not distinct. 2. No valvulae conniventes are seen. 3. Loop-formation is distinct. 4. The color of the affected loop is darker. 5. There is no tendency to stair-step arrangement. 6. The distended bowel has smooth edges. When these findings are present, in association with typical pain, increased tenderness, an increased pulse rate, and a mass, Cole believes that a distinction usually can be made between simple and strangulation obstruction. An increase in muscle rigidity is also useful, although it is a late sign, which may indicate perforation.

All these attempts at differentiation are laudable, and if their objective could be achieved, which is perhaps the major difficulty in determining the management of each special case of intestinal obstruction, difficulty in diagnosis would at once be eliminated. Examination of the patients in our series in whom strangulation was present shows, however, that the criteria just listed are not reliable. Some of the signs and symptoms specified were present in every patient, but there was no distinct pattern, and they were also present, with equal frequency, in the patients in whom strangulation did not occur.

Fever, an increase in the pulse rate, and the white blood cell count may be cited in evidence. In the 15 patients in whom resection was required, the temperature ranged from 96.4 F. to 101 F. The range in the patients in whom resection was not required was from 96.4 F. to 102.4 F. In the resected patients the pulse rate ranged from 54 to 130. The range in the patients in whom resection was not required was from 60 to 160. In the resected patients the white blood cell count ranged from 6,000 to 30,300 per cu. mm. and was above 15,000 per cu. mm. in only 3 of the 15 patients. In the 35 nonresected patients it ranged from 4,900 to 30,000 per cu. mm. and was above 15,000 per cu. mm. in 8 patients. There is no precise diagnostic help in any of these observations.

Our own belief is that no absolute criteria exist by which simple small bowel obstruction can be definitely distinguished from the strangulated variety. The only practical plan is to explore promptly every patient in whom the diagnosis is not absolutely clear-cut. Even when that principle is followed errors will occur and they may be fatal.

The impossibility of being certain that strangulation is not present is one reason why we oppose any therapy for intussusception except surgery, in the form of simple reduction, when the bowel is viable, and resection when it is not. The 2 cases of intussusception in this series occurred in young children, as is usual, but the 4 month old boy who had been ill for 48 hours and who was rated

as a poor risk required only reduction, while the 3 year old girl who had been ill only eight hours and was rated as a good risk on admission required resection of 25 cm. of small bowel. For that necessity we were responsible. Her good condition on admission misled us and she was treated conservatively for 48 hours. A hemangioma, which apparently had initiated the intussusception, was excised with a gangrenous portion of bowel. As Kahle and Thompson[7] have pointed out in an excellent study of 71 cases of intussusception in very young children, it is not always possible, even when the abdomen is opened, to be absolutely certain that vascular changes have not occurred in the bowel. In 1 patient in their series, in whom reduction was obtained by diagnostic barium enema, exploration was done to verify the status of the intestine. There seemed no doubt of its viability at the time, yet death occurred nine hours later, and autopsy revealed gangrene of the affected segment, with peritonitis.

MORTALITY

The lethal factors in acute obstruction of the small bowel are well established. They are physiologic imbalance, strangulation, and prolonged distention, which leads to toxic absorption and alters the permeability of the intestinal mucosa. The role of the unidentified pigment found in the peritoneal fluid by Cohn[4] and his associates remains to be clarified; experimentally it is lethal.

According to Dennis,[5] the mortality rate in the period before Wangensteen popularized the technic of intestinal decompression was in the neighborhood of 40 per cent, against 11 per cent at the present time. If the latter figure be accepted as correct, the mortality rate in this series—2 of 50 cases or 4 per cent—is creditable. One of the deaths, however, as has already been pointed out, should not have occurred. The patient was treated too long by intubation and came to operation as a poor risk instead of a reasonably good risk when she first was seen. In the other fatality, we must take the responsibility for the death, but surely it goes back to the deplorable practice—which in this instance was due to acute appendicitis—of treating abdominal pain by cathartics and chemotherapy in the absence of a definite diagnosis.

SUMMARY

The 50 cases of acute obstruction of the small bowel which occurred at the Lutheran Hospital in Vicksburg over the 7 year period ending in 1953 resulted in two deaths, one of which occurred because of an error of judgment in treating the patient for too long a time by intestinal decompression.

The methods which have done most to reduce the mortality rate of this condition, namely, adjunct therapy, antibiotic therapy, and intestinal decompression, are responsible, because of their unwise use, for many deaths still associated with acute obstruction of the small bowel.

Prompt surgery remains the only sound method of management, the operation employed depending upon the pathologic process present and the status of the bowel. Surgery should be employed with equal promptness if there is any doubt at all of the diagnosis.

The various criteria set up for distinguishing between simple and strangulation obstruction theoretically are sound but practically unreliable.

The 50 cases which make up this series represent a cross section of general surgical practice from the standpoint of etiologic origin, race and sex. The majority of the patients were over 40 years of age. The consideration of most importance in this analysis is that the cases were managed in a small general hospital, in a small community, in which, although some concessions must sometimes be made for the sake of expediency, there is never any compromise with fundamental surgical principles.

REFERENCES

1. Becker, W. F.: Acute adhesive ileus; study of 412 cases with particular reference to abuse of tube decompression in treatment, Surg., Gynec. & Obst. *95:* 472 (Oct.) 1952.
2. Bollinger, J. A., and Fowler, E. F.: Results of treatment of acute small bowel obstruction; clinical study of two hundred five consecutive cases, A. M. A. Arch. Surg. *66:* 888 (June) 1953.
3. Boyce, F. F., and McFetridge, E. M.: Acute intestinal obstruction; comparative analysis of 3 series totaling 715 cases, South. Surgeon *6:* 109 (April) 1937.
4. Cohn, I., Jr.: Strangulation obstruction; recent experimental work, J. Louisiana M. Soc. *105:* 344 (Sept.) 1953.
5. Dennis, C.: Current procedure in management of obstruction of small intestine, J. A. M. A. *154:* 463 (Feb. 6) 1954.
6. Evans, E. I., and Bigger, I. A.: Early recognition and management of intestinal strangulation, J. A. M. A. *133:* 513 (Feb. 22) 1947.
7. Kahle, H. R., and Thompson, C. T.: Diagnostic and therapeutic considerations of intussusception, Surg., Gynec. & Obst. *97:* 693 (Dec.) 1953.

SURFACE CECOSTOMY AS A PROCEDURE FOR THE DECOMPRESSION OF THE ACUTELY OBSTRUCTIVE COLON

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Acute obstruction of the colon is reportedly uncommon; however, my colleagues and I have had a sufficient number of cases to form certain opinions and to standardize an effective technic for decompression.

In large clinics to which patients are brought from afar the incidence of acute colon obstruction is low, while in congested centers it rises materially. The incidence as reported by Rankin[9] was 5 per cent, Gregg and Dixon[7] 5.5 per cent, Rea, Smith and Schwyzer[10] 15.2 per cent, Campbell[14] over 30 per cent, Burgess[1] 35.6 per cent, Graham[6] 16 per cent, Brindley[2] 20.6 per cent (190 cases), Dennis[5] (University of Minnesota, 367 cases) 9.54 per cent and Rosser[11] 10 per cent (private patients) and 20 per cent (clinic). The incidence in our series approaches these figures, there being 12 cases of acute obstruction and 8 of moderate obstruction in 100 cases of cancer of the colon, in which a surgical decompression was done within two days after the patients entered the hospital.

MORTALITY RATE

The operative mortality rate from surgical decompression of the proximal distended colon is excessive. Bruusgaard's[3] report of 13 deaths in 91 collected cases of volvulus is high but not too excessive when it is considered that a volvulus often requires resection.

Brindley[2] reported seven deaths in 34 cases of acute malignant obstruction, a mortality rate of 20.6 per cent, and Gregg and Dixon[7] reported a rate of 34.7 per cent in 121 cases. Mitchell and McCafferty[8] report a mortality rate of 29.1 per cent in 103 cases of all types of colon obstruction and 32.4 per cent in 74 cases of acute malignant obstruction. We have four deaths in 15 cases, 26.6 per cent, and two deaths in 30 cases decompressed by our present method using our special colostomy clamp. One died on the fifth postoperative day from a coronary occlusion and 1 after cecostomy six days following mid-colon resection with an associated gastric malignancy that, because of extensive cecal distention, required cecostomy. Peritonitis was not found at autopsy.

FACTORS CONTRIBUTING TO MORTALITY

Factors relating to mortality are those pertaining to the patient and those concerned with errors in surgical judgment or with faulty technic. Those pertaining to the patient are related to age, nutrition and serious associated disease. Age, nutritional changes and many associated diseases often can be compensated

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for, except in sudden emergencies, when time does not permit appropriate preparation. Errors in judgment of the surgeon are factors contributing to mortality. They are related to failure to regard obstruction of the large bowel as separate and distinct from obstruction of the small bowel, delay in decompression, inadvisable abdominal exploration and ill advised primary resection. Technical errors are related to unnecessary trauma, manipulation, spillage and suture of the bowel to the abdominal wall.

ROENTGENOLOGIC STUDY

Roentgenologic study does not give as much information in the obstructed colon as in obstruction of the small bowel. It does show a distended proximal colon with its characteristic haustral markings, but the exact site of the obstruction often is obscure. Localization of the lesion can be made by means of a barium enema, if the obstruction is complete. The barium is no obstacle to surgical intervention since it readily can be evacuated by one or more enemas. It is only when the obstruction is incomplete that subsequent difficulty may be encountered after a barium enema. The proximal colon may become impacted with dehydrated barium, which may convert an incomplete obstruction into a complete one. Barium rarely, if ever, should be given by mouth in suspected obstruction of either the small or the large bowel.

The pattern assumed by a distended colon is one that conforms to the anatomic position of the colon and usually it shows the characteristic haustral markings. As the colon becomes more distended because of the closed loop nature of the obstruction, these characteristic haustral markings are not so well seen. These are fine points of roentgenologic interpretation and are easily detected by a competent radiologist.

When the ileocecal valve is competent there is a double obstruction in which the blood supply soon becomes inadequate to maintain bowel viability throughout its entirety. The segment of the colon most distensible is the cecum, the wall of which becomes extremely thin and ultimately small areas become gangrenous and perforate. This closed loop type of colonic obstruction is in part comparable to the strangulated or volvulus type of obstruction of the small bowel. It is in reality as much of an emergency as strangulation of the small bowel. Therefore, surgical decompression should not be long deferred, else gangrene and rupture of the cecum will occur. We have observed this twice. In one instance it occurred while abdominal preparation was being made for decompression of the colon. A cecostomy was immediately made and fortunately there was little abdominal soiling and recovery occurred. Subsequent resection of the primary lesion was done successfully.

SURGICAL PRINCIPLES

Obstruction of the colon presents a complication which prohibits a primary attack on the lesion, which is in contrast to obstruction of the small bowel. In the former, the problem is to decompress the colon and prepare it for subsequent resection of the lesion, while in obstruction of the small bowel surgical

intervention is directed primarily at removal of the lesion. It is not only in acute obstruction of the colon that decompression is desired, but in all cases of chronic obstruction of the left side of the colon preliminary decompression of the proximal portion of the bowel is indicated. Only on a clean, nonobstructed colon can primary resection and anastomosis be done safely.

Obstructive lesions of the right side of the colon may be decompressed by means of cecostomy with or without ileocolostomy. Ileocolostomy is desirable and sufficient when the lesion is fixed and inflammatory and the ileocecal valve is incompetent. Lesions in the left side of the colon usually are annular and constricting in character and at the time of consultation there frequently is found a variable degree of obstruction and in a few cases complete obstruction.

Fortunately, most obstructions are not actually complete but are in part due to edema, inflammatory reaction and invaginated mucous membrane. With proper surgical decompression and by means of irrigations, the obstruction will relent sufficiently to permit the colon to empty itself partially through the distal segment. This materially aids the thorough cleansing of the colon.

Cecostomy as the preferred measure of decompression of the acutely obstructed colon is controversial. Cecostomy as it usually is done, with a catheter held with a purse string in the cecum and the cecal wall sutured to the parietal peritoneum functions only as a vent for the escape of gas. Irrigations and bowel preparation cannot be done through a small opening in the cecum fixed to the parietal peritoneum and connected to the outside by a fecal fistula. It is not a cecostomy—it is a cecal fecal fistula. A similar technic used upon the right colon for the same reason would be equally ineffective and would be in like manner a colon fecal fistula. It would be of no service as an avenue to irrigate and clean the bowel. A cecostomy that is comparable to a colostomy with a large outside stoma, as a colostomy has, is adequate for thorough irrigation and is much easier subsequently to close. The lumen is large, the approach is easy, omental attachments are not present and edema and induration will not be sufficient to obstruct the lumen, as may occur in colostomy closure.

Our cecostomies are made as a colostomy is made with a large cone of cecum delivered to the outside which provides an adequate external opening for drainage and for irrigation. No suturing of the bowel to the peritoneum or the abdominal wall is done. The bowel readily adheres to these structures. Edema of the protruding segment of cecum in a few hours prevents it from retracting into the abdomen. Our cecostomy clamp retains it outside until this swelling and edema occurs. The clamp is removed in 48 to 72 hours*. This is truly a functioning type of cecostomy and has advantages comparable to a colostomy and in many instances superior.

We believe this type of cecostomy to be the best procedure for decompression of the acutely obstructed colon for the following reasons:

First—In acute obstruction of the colon the most distendible part of the colon is the cecum. It is the segment that in time may perforate. This will occur when the intracecal pressure becomes greater than the systolic blood pressure.

* Clamp is made by V. Mueller & Co., Chicago.

The blood then cannot be carried to the antimesenteric region of the cecum and areas of necrosis will develop. Perforation will eventually occur.

Second—A surface cecostomy with a good stomal opening permits colon decompression and releases the intracolonic pressure at the site of the neoplastic obstruction. Edema and induration subside and the bowel at the site of the neoplasm again becomes partly patent. With the release of this associated agent of obstruction, edema and induration, the bowel may be irrigated and thoroughly cleaned by enema and cecal irrigations. In no instance is it impossible to thoroughly cleanse the bowel and prepare it for satisfactory subsequent resection when the cecostomy has a good open stoma presenting upon the surface of the abdomen.

Third—The distended cecum is the most accessible segment of the large bowel for surgical decompression. It is the most distendable portion of the obstructed colon. It in no way interferes with an extensive resection—often required for lesions of the left colon.

Fourth—Decompression of the right colon adds little to the solid character of the fecal discharge and contributes only little to the thoroughness of subsequent colon irrigations and cleansing of the lower colon. The mechanics are the same—the colon is equally decompressed and the edema and induration at the obstructive site will subside, patency of the bowel will again be re-established and through and through irrigation from rectum to cecostomy opening can be accomplished as well as it can with a right colon colostomy, if the stomas are adequate in size.

Fifth—The right colon is hard to approach surgically and decompress when distended. It delivers poorly due to omental and mesenteric attachments. A glass rod under the distended colon may, through pressure, erode into a vessel and produce bleeding, and a distended right colon is difficult to deliver into the field of operation for such support and for tubal decompression.

Sixth—A right colostomy imparts no information relative to the viability of the cecum which may contain areas of devitalization which are unknown to the surgeon at the time of right colostomy. If present they will perforate even though a successful right colostomy has been done. This we have seen on three occasions. Most undesirable is the fact that a right colostomy materially interferes with the extensive mobilization often necessary for radical resection of left colon lesions. Left colectomy with contemplated anastomosis of the left transverse colon to the lower sigmoid or recto-sigmoid is difficult or impossible to accomplish when a right colostomy is present. This procedure more and more is being advocated as it is frequently surgically indicated for radical extirpation of gland bearing tissue. In such instances it usually is necessary to mobilize the hepatic flexure for adequate anastomosis without tension. This cannot be done when a right colostomy is present. This can be done easily with a cecostomy. Why then handicap future surgical procedures by a colon decompression which restricts extensive resection, mobilization and secure anastomosis? Especially is this objectionable when decompression and adequate colon preparation for subsequent surgery can be done by a well performed surface cecostomy.

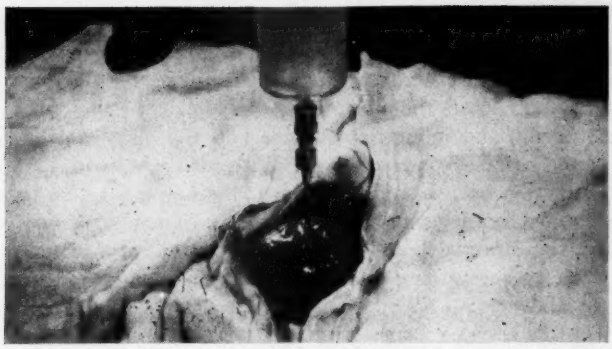

FIG. 1. Shows method of deflating cecum by needle puncture

We, therefore, believe a cecostomy with an adequate skin surface stoma is preferable to colostomy for decompression of the acutely obstructed left colon due to intrinsic malignant disease.

ADVOCATED PROCEDURE FOR DECOMPRESSION OF THE COLON

Through an adequate incision under pentothal or spinal anesthesia (not local anesthesia) we deflate the distended cecum or colon by needle puncture (fig. 1) and grasp the deflated bowel at the point of needle puncture with a flat—no tooth—thumb forcep (fig. 2). Our special clamp (fig. 3) is seen applied to an adequately suspended cone of bowel. The abdomen has been closed completely around this protruding segment of cecum or colon before it is opened to insert a catheter into the bowel through the round opening in the clamp. Figures 4 and 5 show the procedure of opening the bowel and the insertion of the catheter into bowel through the hole in the clamp. The catheter is fixed by suture to prevent its slipping out. This procedure does not completely divert the fecal current. It is advocated only for an acute obstruction of the colon due to malignant disease for which resection is subsequently contemplated.

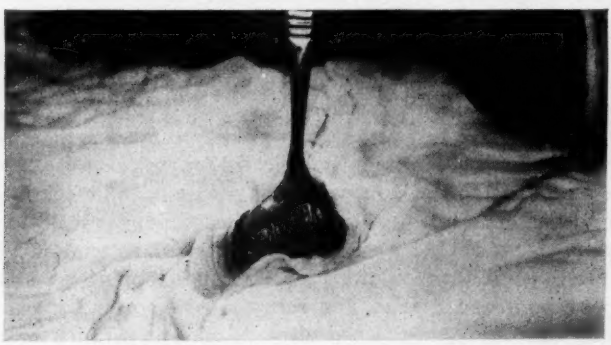

FIG. 2. The deflated bowel is clasped at the point of needle puncture with a flat no tooth thumb forcep.

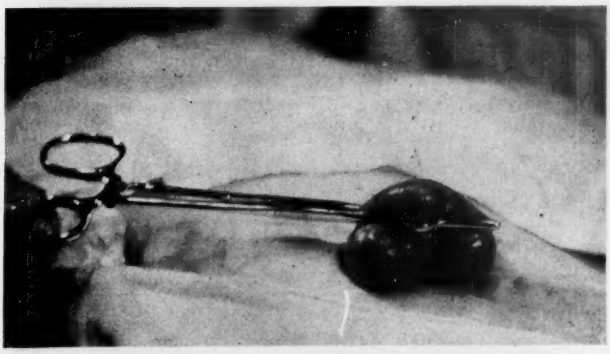

FIG. 3. Shows the cecostomy clamp applied to a wide segment of the exteriorized bowel. The abdomen is closed before the bowel is opened for insertion of catheter.

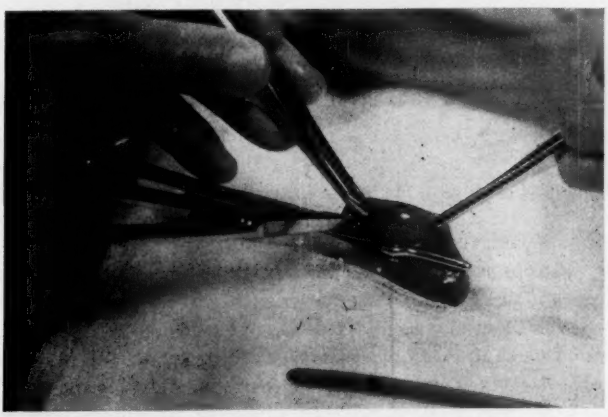

FIG. 4. Shows segment of bowel suspended by thumb forceps for opening and insertion of catheter.

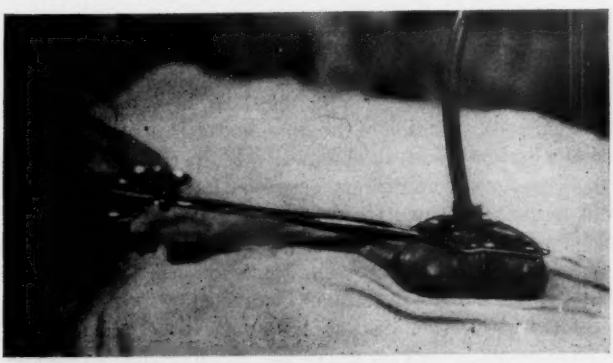

FIG. 5. Shows catheter in lumen of bowel through hole in clamp. A wide stoma will result after catheter is removed.

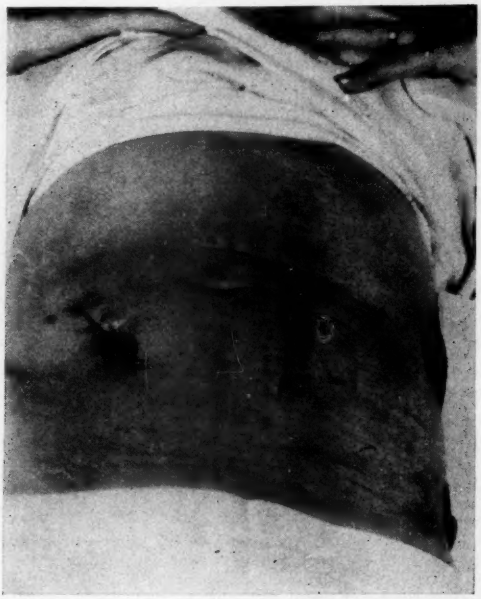

FIG. 6. Shows size of stoma a few weeks later after second operation has been done

This method exteriorizes only an adequate segment of the cecum or colon and gives a sufficient vent for gas to pass through the catheter. It makes a good skin surface stoma in either instance for irrigation.

The inserted catheter acts as a vent largerly for gas and prevents redistention of the bowel. The clamp is removed later, or it sloughs off, leaving a good stoma and the bowel is later irrigated and cleansed (fig. 6). This method is simple, practically aseptic and is done without difficulty. It always should be done under a light anesthesia. Relaxation and not anxiety and abdominal resistance is very essential for bowel exposure and decompression. We have tried both.

SUMMARY

The incidence and mortality rate of acute obstruction of the colon are discussed and statistics are reported. The comparable relationship of obstruction of the colon to strangulated obstruction of the small bowel is mentioned.

The value of a barium enema in obstruction of the colon and its complications are stressed. Sites for decompression based on location of the lesion are emphasized and a special technic for decompression is described. The technical disadvantage of right colostomy for lesions near the upper left colon are discussed. A similar disadvantage may be present for a low left lesion where extensive resection and mobilization are likely to be demanded.

Tube cecostomy with bowel fixation to the parietal peritoneum is condemned. It is only a fecal fistula, a vent for gas escape and furnishes no avenue for bowel

irrigation. Its technical steps are conducive to spillage, infection and peritonitis. Suture of the distended colon to the parietal peritoneum is unnecessary and presents avenues for contamination and leakage.

A properly made cecostomy with an adequate surface stoma can serve as an avenue for successful bowel irrigation. Cecostomy by the method described is easily done. It is extra-abdominal and fills all the requirements for decompression and bowel irrigation. No sutures are necessary. The cecum soon will adhere to the abdominal wall.

REFERENCES

1. Barling, S., et al in discussion on Burgess, A. H.: Treatment of obstruction of colon, Brit. M. J. *2:* 547 (Sept. 29) 1923.
2. Brindley, G. V.: Acute obstructions of colon, Texas State J. Med. *40:* 571 (March) 1945.
3. Bruusgaard, C.: Volvulus of sigmoid colon and its treatment, Surgery *22:* 466 (Sept.) 1947.
4. Campbell, O. J.: Surgery of carcinoma of colon, Ann. Surg. *94:* 705 (March) 1931.
5. Dennis, C.: Treatment of large bowel obstruction; transverse colostomy-incidence of incompetency of ileocecal valve; experience at University of Minnesota Hospitals, Surgery *15:* 713 (May) 1944.
6. Graham, R. R.: Carcinoma of colon, Am. J. Digest Dis. and Nutrition *1:* 584 (Oct.) 1934.
7. Gregg, R. O., and Dixon, C. F.: Operative malignant lesions of colon producing obstruction, S. Clin. North America *21:* 1143 (Aug.) 1941.
8. Michel, M. L., and McCafferty, E. L., Jr.: Acute obstruction of colon with special reference to factors of mortality, Arch. Surg. *57:* 774 (Dec.) 1948.
9. Rankin, F. W.: Resection and obstruction of colon (obstructive resection), Surg., Gynec. & Obst. *50:* 594 (March) 1930.
10. Rea, C. E., Smith, B. A., Jr., and Schwyser, H.: Carcinoma of colon and rectum, Bull. Staff Meeting, Minnesota General Hospital; *354:* 364 (April 28) 1939.
11. Rosser, C.: Personal communication to author.

CURRENT MANAGEMENT OF BENIGN AND MALIGNANT PANCREATIC TUMORS

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Benign and malignant tumors of the pancreas and the periampullary region are not extremely common but neither are they rare. They produce a wide variety of symptoms and, because of the profound physiologic and metabolic disturbances that may attend them, their early recognition and proper treatment are important.

A simple classification which embraces only the commoner types of pancreatic tumors is sufficiently comprehensive to encompass one's clinical experience.

CLASSIFICATION

Benign Tumors

1. Pancreatic heterotopia
2. Cystadenoma
3. Benign islet cell adenoma

Malignant Tumors

1. Carcinoma of the head
2. Carcinoma of the body and tail
3. Carcinoma of the ampulla of Vater
4. Cystadenocarcinoma
5. Islet cell carcinoma

PANCREATIC HETEROTOPIA

Ectopic or aberrant pancreatic tissue may occur at any level along the gastrointestinal tract but is commonest in the region of the pylorus and duodenum. These pancreatic rests have a certain propensity to occur in diverticula along the gastrointestinal tract, as is reflected by the fact that in the large series observed by Barbosa, Dockerty and Waugh,[2] 5.3 per cent of the group occurred in Meckel's diverticulum.

These heterotopic masses may be only loosely adherent to the serosal surface of the intestine or may appear in the subserosal, submuscularis or submucosal position.

For the most part they cause little or no symptomatic disturbance. They may, on the other hand, produce disturbances in motility and, when they are submucosal, they may cause disturbances in secretory activity and ulceration. They occasionally cause active bleeding. When they occur within the wall of the small intestine they produce more severe motor disturbances, and if they become sufficiently large they may incite intussusception. This is particularly true when the pancreatic heterotopic mass occurs in a Meckel's diverticulum.

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Some of these pancreatic heterotopic masses exhibit physiologic activity in both acinar and islet cell tissue. Hyperinsulinism has been reported in some instances in which the ectopic pancreatic mass contained an islet cell adenoma. Carcinomas also have been reported arising in these aberrant pancreatic masses.

Marshall and Curtiss[6] have reported from this clinic 5 instances of ectopic pancreatic tissue involving the stomach and associated with significant symptoms. These authors also described 2 cases of aberrant pancreas of the duodenum in which epigastric pain occurred. In 3 of these patients with pancreatic masses involving the stomach and duodenum active ulceration overlying the tumor was present.

Treatment. Local excision is adequate in the treatment of these lesions provided no other associated disease is present which dictates more radical therapy. These tumors, however, are frequently mistaken for more serious types of lesions, particularly when there is ulceration overlying them, and this misinterpretation may lead to subtotal gastric resection. If ulceration with bleeding has occurred, it is perhaps wise to do a more radical procedure even though the pancreatic heterotopic mass is properly recognized.

If intussusception or intestinal obstruction arises from these aberrant pancreatic masses, local resection should be done.

CYSTADENOMAS

Cystadenomas of the pancreas are extremely rare. In a previous series of cysts of the pancreas reported from this clinic, Cattell and Warren[3] observed only one papillary cystadenoma. These tumors are characterized by their irregular lobulation and the variable consistency from one part of the tumor to another. The fluid contents of the multiple loculi are also variable, some showing serous fluid while others show serosanguinous or mucoid material. The papillary projections may also be confined to one or a small number of the total cavities. These tumors may grow very slowly and may in rare instances undergo malignant degeneration.

These cysts should be treated by partial pancreatectomy, removing the segment of involved pancreas. Because of the variation from one cavity to another it is possible to overlook the papillary nature of these tumors and to treat them by some form of external or internal drainage. The presence of a neoplastic cyst always should be suspected when the exposed surface is lobulated and when there is little or no peripancreatitis.

CYSTADENOCARCINOMA

It is a minor paradox in the pathology of pancreatic tumors to observe that, whereas cystadenomas are extremely uncommon, cystadenocarcinomas are more frequently observed. In the first 46 cases of pancreatic cysts analyzed at the Lahey Clinic, there were 5 instances of cystadenocarcinomas as compared with one cystadenoma. These malignant tumors, like the benign cystadenomas, are slow-growing, multilocular, frequently irregular in outline and of relatively low grade malignancy. They usually produce minimal symptoms early in their course and only after they have attained sufficient size to exert pressure on the

surrounding structures do they cause any significant disturbance. Two of the 5 patients with cystadenocarcinoma treated at the clinic had been operated upon previously elsewhere, with external drainage done in each instance and without the malignant nature of the tumor having been recognized at the original procedure. Papillary cystadenocarcinomas of the pancreas may attain considerable size but, despite this tendency, they usually can be completely extirpated. Again, a note of warning should be sounded against the internal drainage or simple marsupialization of this type of cyst. Internal drainage should be reserved for those papillary cystadenocarcinomas that cannot be extirpated surgically. In the series of 5 patients observed at the clinic one cyst was drained into the jejunum because it could not be removed, three were excised by distal pancreatectomy and one was removed by total pancreatectomy since it involved most of the gland. All 4 patients, in whom it was possible to remove the tumor, are alive and without obvious recurrence, ranging from 18 months to 4 years. The encouraging results in those malignant tumors that were excised indicate the validity of an aggressive attitude toward papillary cystadenocarcinoma of the pancreas.

ISLET CELL ADENOMAS AND ISLET CELL CARCINOMAS

Tumors arising from the islet cells constitute one of the most interesting chapters in the field of pancreatic surgery. These tumors may be either benign or malignant, they may exhibit physiologic activity characterized by hyperinsulinism or be nonfunctioning tumors. The hyperinsulinism may appear in either the benign or the malignant series, and indeed metastatic growths from malignant islet cell tumors often share in this physiologic activity. It is frequently difficult to determine by microscopic analysis whether a given islet cell tumor is malignant or benign. This fact has been responsible for placing some of these tumors into a tentative group of presumably benign tumors. Duff,[5] who has analyzed a large group of these tumors, has pointed out that, although some of them show microscopic evidence of capsular and blood vessel invasion, many in this particular group have been followed for a prolonged time without evidence of local recurrence or distant metastases.

It has not yet been possible to detect precisely why some of these tumors exhibit hyperinsulinism while others are nonfunctioning in this regard. It is known, however, that the degree of hyperinsulinism does not depend necessarily upon the size of the tumor. Extreme hyperinsulinism may appear in a tumor that measures not more than 2.5 mm. in diameter. Large adenomas, on the other hand, may appear without any hyperinsulinism. We have observed one nonfunctioning islet cell carcinoma approximately 2 inches in diameter in an 11 year old child.

Hyperinsulinism. The symptoms associated with hyperfunctioning islet cell tumors depend upon the excessive secretion of insulin which in turn results in hypoglycemia, and the ultimate decrease below the critical functional level in the supply of glucose to the brain. The more dramatic symptoms of hyperinsulinism are manifestations of hypoglycemia causing decreased cerebral metabolism.

Clinical Manifestations. The symptomatology of hyperinsulinism includes psychic manifestations such as restlessness, anxiety, confusion, coma and transient neurologic manifestations and convulsions. Disturbances in the sympathetic-parasympathetic system result in nausea, sweating, visual disturbances and fainting.

Attacks are usually preceded by a period of fasting and are particularly prone to occur during severe muscular exercise after a prolonged fast. The attack is frequently preceded by extreme hunger and an acute awareness that some physiologic crisis is at hand. Agitation, perspiration, pallor, dizziness and weakness usually follow rapidly upon the prodromal manifestations. Severe attacks are then characterized by tonic and clonic convulsions and deep coma.

These patients learn in time that they may ward off an attack or minimize it by the ingestion of food, particularly by rapidly absorbable sugar. They develop, more or less unconsciously, the habit of excessive ingestion of carbohydrates and may gain considerable weight even in the presence of a malignant tumor.

Whipple[8, 9] set forth the classical triad upon which the clinical diagnosis is based. These include: 1. The attacks must come on during periods of fasting or after extreme exercise. 2. The blood sugar value during an attack or after prolonged fasting must be below 50 mg. per 100 cc. 3. The symptoms must be relieved promptly by oral or intravenous administration of sugar. It is also wise in approaching the diagnosis to insist, as emphasized by Allan,[1] that the symptoms must be severe enough to cause stupor or loss of consciousness, that diseases outside of the pancreas capable of producing hypoglycemia must be excluded, and that the self administration of insulin must be ruled out. If these criteria are insisted upon in studying patients suspected of having hyperinsulinism, an adenoma or an islet cell carcinoma will be found in a high percentage of cases.

Surgical Treatment. Once it has been demonstrated that the patient's clinical manifestations and laboratory findings are characteristic of hyperinsulinism, surgical exploration should be carried out. These tumors are frequently small. They may appear in any location within the pancreas and their detection may require complete mobilization of the entire tail and body of the gland and wide mobilization of the head as well. In searching for an islet cell adenoma, sight should not be lost of the fact that in approximately 2 per cent of instances the islet cell tumor will be in an ectopic mass.

Once the tumor has been found it should be removed by enucleation if a well-circumscribed encapsulated tumor is present. If there is any suggestion of local invasion, partial pancreatectomy should be done. This will usually mean resection of the tail and body of the gland, but occasionally an islet cell adenoma of the head may be observed which requires pancreatoduodenal resection. This procedure should not be employed, however, unless it can be demonstrated that the tumor is malignant, since the hazard of this procedure is too great to accept for the treatment of a benign lesion. Since the incidence of cancer in these tumors is high, all should be submitted to immediate frozen section analysis in order that more radical resection can be done if malignancy is observed.

Total pancreatectomy has been done for hyperinsulinism in 2 instances at the Mayo Clinic. The results of these procedures have been reported by Priestley,

Comfort and Radcliffe.[7] Total pancreatectomy should not be employed until all of the means of detecting the adenoma have been exhausted and until less heroic methods of attempting to relieve the hyperinsulinism have been undertaken.

In 14 patients who were operated upon and observed at the Lahey Clinic, local excision was done in 5, distal pancreatectomy with removal of a demonstrable tumor in 5, distal pancreatectomy without demonstrable islet cell tumors was done in 2 instances of hyperinsulinism and pancreatoduodenal resection was done in 2 patients.

In a large series of collected cases in which operation was done for hyperinsulinism, an adenoma was found in 65 to 75 per cent of patients. Multiple adenomas were present in 12 per cent. A hyperfunctioning adenoma was present in an ectopic mass in 2 per cent. An islet cell carcinoma with obvious metastases was demonstrable in 10 per cent and two-thirds of this group with obviously malignant tumors showed hyperinsulinism. In about 20 per cent of cases the tumor is frankly or questionably malignant. In 25 per cent of patients who have exploratory operations for hyperinsulinism, no adenoma is found. Six of the 14 patients operated upon at the clinic had malignant tumors. Two of these were nonfunctioning islet cell carcinomas arising in the head necessitating pancreatoduodenal resection. One of these patients survived approximately five years and died of recurrence of the carcinoma. The other malignant tumor treated by pancreatoduodenal resection occurred in an 11 year old girl who is now alive and well one year after resection. One of the remaining patients who exhibited malignant disease died 18 months later from recurrence; the other 3 are alive and well from one to five years.

CARCINOMA OF THE PANCREAS AND PERIAMPULLARY AREA

Carcinomas arising in the head of the pancreas, the ampulla of Vater, the duodenum and the distal common duct have many similarities both in terms of clinical manifestations and of surgical requirements for their relief. The common denominator of their symptoms is jaundice. It is frequently possible to distinguish on a clinical basis alone, as previously emphasized by Cattell, between a carcinoma arising in the head of the pancreas and a carcinoma of the ampulla of Vater. It is more difficult, but sometimes possible, to recognize carcinoma arising in the distal common bile duct and in the duodenum on the basis of clinical data.

The classical concept that carcinoma of the head of the pancreas and ampulla of Vater is characterized primarily by painless jaundice is not valid. A certain degree of pain is usually present in both conditions, but is almost universal in carcinoma of the head of the pancreas while it may be absent in a significant percentage of cases of carcinoma of the ampulla. The outstanding symptoms are pain, weight loss, jaundice, fatigue, anorexia, diarrhea and constipation.

In general, carcinoma of the head of the pancreas has an insidious onset marked by anorexia, fatigue, loss of weight, pain and then jaundice. Disturbances in digestion, frequently characterized by diarrhea, are likewise common. In carcinoma of the ampulla of Vater jaundice is an early symptom and its onset usually precedes any marked loss of weight or fatigue.

Important physical findings include jaundice, enlarged liver and evidence of weight loss. A palpable gallbladder can be felt in only 25 to 45 per cent of the patients although it is distended (when present) as observed at the operating table in 75 to 80 per cent of cases. A palpable tumor is rarely observed in resectable cases.

Carcinoma of the duodenum usually is marked by a longer history of physical deterioration, by chronic indigestion and marked secondary anemia.

The demonstration of increased bilirubinemia and elevated alkaline phosphatase without marked disturbance in liver function helps in the diagnosis. Steatorrhea and creatorrhea are more common in cases of carcinoma of the head of the pancreas since the duct of Wirsung is obstructed in most instances, whereas in carcinoma of the ampulla of Vāter the duct of Wirsung is obstructed in only about 20 per cent of cases.

Roentgenologic findings include enlargement of the duodenal sweep, irregularity of the duodenal silhouette, particularly on the medial border, by erosion around the papillae, especially in the presence of ampullary tumors, and, occasionally, by complete obstruction of the duodenum. Primary duodenal tumors exhibit characteristic filling defects, usually with ulceration.

Treatment. Carcinomas arising in the head of the pancreas and the periampullary area are suitable for resection if they are confined to the region that can be safely circumscribed surgically. Lesions arising in the distal common bile duct and the ampulla of Vater and duodenum are more favorable from the standpoint of resection than are carcinomas arising in the head. Tumors arising in the head of the pancreas tend to invade surrounding tissue and to spread by lymphatic and perineural channels and are for this reason frequently unfavorable for resection. Because of the poor over-all results of pancreatoduodenal resection for carcinoma of the head, particular care should be taken to search for regional or distant metastases before proceeding with the radical resection.

In 88 cases of carcinoma arising in the head of the pancreas and periampullary area previously reported from the clinic by Cattell and Warren,[4] 5 arose in the distal common bile duct, 7 in the duodenum, 30 in the ampulla of Vater, and 46 in the head of the pancreas.

One of the most important single technical considerations in resection of the head of the pancreas is restoration of the continuity of the pancreas with the intestinal tract, which we now prefer to do in every instance by direct anastomosis of the duct of Wirsung to a loop of jejunum. In the presence of prolonged, severe jaundice with marked liver damage we still prefer, in this clinic, to do the operation in two stages. At the present time, approximately 80 per cent of the resections are done in one stage.

Mortality. The mortality rate of pancreatoduodenal resection (including two total pancreatectomies) for malignant lesions in this series was 13.6 per cent, with the mortality rate of resection for carcinoma of the ampulla of Vater being 6.6 per cent and carcinoma of the head of the pancreas 17.3 per cent. The current mortality rate for pancreatoduodenal resection at the clinic is about 8 per cent.

Results. Approximately 30 per cent of patients surviving resection for carcinoma arising in the ampulla of Vater lived five years and 3 of these patients are alive

and well and free of recurrence more than 10 years after resection. The survival rate following resection for carcinoma of the head of the pancreas, based upon an analysis of those cases in which five years or more have elapsed since resection, was 12 per cent.

Since the prognosis for carcinoma of the ampulla of Vater following pancreatoduodenal resection is so much better than for tumors arising in the head of the pancreas, great care should be taken at the time of exploration to distinguish between tumors arising in these two particular areas. We have observed 3 patients at the clinic, 1 operated upon 14 months, another 18 months and another three years before exploratory operation at the clinic, each of whom had carcinoma arising in the ampulla of Vater, and in each instance the tumor was still resectable at the time they were operated upon at the clinic. Each of these patients survived 18 months following resection and 1 is alive but has obvious recurrence more than two years after resection. Failure to distinguish between carcinoma arising in the ampulla of Vater and carcinoma of the head undoubtedly has accounted for some needless mortality in this field.

Although there is less room for optimism regarding resection of tumors arising in the head of the pancreas, this procedure should not be completely abandoned. The need is for earlier detection and for more precise appraisal at the time of the operation with respect to operability.

Attempts to salvage some of these patients by the radical extension of the scope of surgery do not seem feasible.

SUMMARY

Tumors of the pancreas are not rare.

The diagnosis frequently is missed because due consideration is not given to the pancreas in the differential diagnosis.

Results of surgical treatment of pancreatic tumors are recorded.

REFERENCES

1. Allan, F. N.: Diagnosis and treatment of hyperinsulinism, S. Clin. North America *15:* 1481 (Dec.) 1935.
2. Barbosa, J. J. deC., Dockerty, M. B., and Waugh, J. M.: Pancreatic heterotopia; review of literature and report of 41 authenticated surgical cases, of which 25 were clinically significant, Surg., Gynec. & Obst. *82:* 527 (May) 1946.
3. Cattell, R. B., and Warren, K. W.: Surgery of the Pancreas, Philadelphia, W. B. Saunders Company, 1953, p 157.
4. Cattell, R. B., and Warren, K. W.: Same as reference 3, p. 327.
5. Duff, G. L.: Pathology of islet cell tumors of pancreas, Am. J. M. Sc. *203:* 437 (March) 1942.
6. Marshall, S. F., and Curtiss, F. M.: Aberrant pancreas in stomach wall, S. Clin. North America *32:* 867 (June) 1952.
7. Priestley, J. T., Comfort, M. W., and Radcliffe, J., Jr.: Total pancreatectomy for hyperinsulinism due to islet-cell adenoma; survival and cure at 16 months after operation; presentation of metabolic studies, Ann. Surg. *119:* 211 (Feb.) 1944.
8. Whipple, A. O.: Surgical therapy of hyperinsulinism, J. internat. chir. *3:* 237, 1938.
9. Whipple, A. O., and Frantz, V. K.: Adenoma of islet cells with hyperinsulinism; a review, Ann. Surg. *101:* 1299 (June) 1935.

THE SURGICAL TREATMENT OF PRIMARY AND SECONDARY HEPATIC MALIGNANT TUMORS

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The feasibility of operating upon the liver is amply demonstrated by the fact that more than 500 instances of excision of hepatic neoplasms are recorded in the literature. In recent years the number of such reports is increasingly frequent. To be sure, total hepatectomy is incompatible with life but the factor of safety would appear to be large judging from animal experiments (dogs) in which about 80 per cent of the liver could be removed and the animal survive. No observations are available that would indicate 80 per cent of the human liver can be removed and the patient survive, but large portions of the liver can be removed with safety.

It would appear that there are two factors regarded as especially dangerous in connection with surgery of the liver in the past, (1) hemorrhage and (2) infection. When incised, the liver parenchyma oozes blood at first vigorously, but if large vessels are clamped the weeping of blood from the parenchyma soon may be arrested by continued pressure with dry gauze or gauze sponges soaked in hot saline solution. As to infection, the human liver, in contrast to that of the dog, is sterile and therefore there is no greater danger of infection than in other operations upon abdominal viscera.[4]

Small tumors may be excised by encompassing eliptical incisions and sharp dissection. Tumors near a margin of the liver may be removed by excision of a wedge shape portion of the parenchyma to include the growth. In both instances, after the excision, pulsating arteries and bleeding veins are clamped and ligated. Large bile ducts are likewise secured and ligated. The freshly cut surfaces are approximated by interrupted catgut sutures which are placed to include generous portions of liver parenchyma. A cardinal principle of such surgery is the placing of soft rubber drains to the operation sites to facilitate escape of bile should this occur during the immediate postoperative period. It almost always occurs, but the author has never observed a permanent biliary fistula resulting from partial hepatectomy. Neoplasms which bulge outward from one of the margins of the liver and have some sort of pedicle may be removed by transection of the pedicle. The raw surface is compressed by a row of interlocking interrupted mattress sutures. The latter serve for hemostasis and compression of the transected bile ducts.

The entire right or left lobe of the liver may be resected. Two methods are possible. The one, a *guillotine* type of liver lobectomy, consists of a vertical transection of the lobe just to the right or left of the falciform ligament, as the case may

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be, leaving a 1 or 2 centimeter layer ot liver tissue at the base of each lobe. In this type of excision great care must be exercised to secure the large blood vessels and bile ducts as the transection progresses. In regard to the left lobe, the author places the index finger of the left hand on the upper surface of the left lobe just to the right (surgeon's) of the falciform ligament and the left thumb on the under surface. Compression of liver parenchyma between these two digits is exerted prior to incision of the liver itself. As large arteries, veins and bile ducts are encountered during the progressive transection by repeated shallow strokes of the scalpel they are clamped and ligated. The left hepatic vein should be identified and doubly clamped before it is divided and the operation terminated by removal of the left lobe. For the right lobe the same principle may be followed, using the right thumb and index finger applied just to the left (surgeon's) of the falciform ligament. Because the right hepatic vein is quite large, considerable care is to be exercised in securing it prior to transection. Leaving a small pyramidal portion of liver parenchyma of the right upper lobe adjacent to the falciform ligament in order to ligate by transfixation sutures may facilitate right lobectomy without the hazards of exposing the right vein. In total right hepatic lobectomy the gallbladder is also removed en masse with the right lobe. Prior to transection of the base of the right lobe, the cystic duct is exposed, doubly ligated and transected. The cystic artery is also divided and ligated. The gallbladder is left attached to the liver. The caudate lobe of the liver may or may not be removed separately; it has its own group of vessels which must be dealt with.

The other methods of performing right and left hepatic lobectomy are of more recent development. In 1936 Huard and Meyer-May[2] described the procedure of detaching (except for division of left hepatic vein) the left lobe by vertical incision just to the left of the falciform ligament and then isolation and ligation of the left branch of the portal vein, left branch of the hepatic artery and left branch of the common bile duct, and finally division of the left hepatic vein. In regard to the right lobe, Lortat-Jacob and Robert[3] described what they refer to as *controlled* right hepatic lobectomy. This consists of exposure by means of a right thoracoabdominal incision starting over the right eighth rib at its angle, and division of the diaphragm to the vena cava. The round and triangular ligaments of the liver are divided and the liver retracted upward into the thorax. The cystic duct, cystic artery and right branches of the hepatic artery and right hepatic duct are transected. The liver is then luxated downward and forward to the left in order to secure and divide the right hepatic vein or veins. The base of the right lobe of the liver is then cut through just to the right of the falciform ligament. The raw surface is covered with falciform ligament.

A variety of hepatic neoplasms, both primary and secondary, have been excised by the author employing the appropriate procedures. In doing right or left hepatic lobectomy the *guillotine* method described above was employed and has given satisfaction, but the *controlled* methods described also have appeal (Huard and Meyer-May; Lortat-Jacob and Robert).

A summary of the experience to date is presented below with addition of 8 patients to those previously reported[1] by the author:

A. *Primary Benign Tumors*

Patient	*Operation*	*Result*
1. E. J. 53 yr. woman. Massive hemangioma.	Resection of almost entire rt. lobe with neoplasm measuring 25 cm. in greatest diameter & weighing 1650 Gm. together with adherent portion of abdominal wall.	Living & well 3 yrs.
2. Kog. 48 yr. woman. Solitary tumor 3 cm. in diameter protruding above surface of right lobe.	Tumor excised at time a radical hysterectomy & pelvic node excision was performed for carcinoma of corpus. Histologic study of hepatic tumor showed "normal liver and fibrosis." Interpreted as "hamartoma" or extra small aberrant liver lobe.	Living & well 3 mos.

B. *Primary Malignant Tumors*

3. Moult. 41 yr. man. Massive angiosarcoma right lobe.	Partial resection. Operation abandoned because of hemorrhage after removal of 1345 Gm. of tumor tissue.	Lived 2 mos. Neoplasm grew rapidly.
4. M. C. 39 yr. woman. Massive neoplasm thought at first to be angioma, then angiosarcoma.	Resection of entire right lobe tumor measured 17 cm. in greatest diameter. Second operation 2 yrs. later for resection of recurrence 5 cm. in diameter.	Living & at present no evidence of recurrence 2 yrs. 4 mos. after first operation.
5. M. Mov. 51 yr. woman. Choleangiocarcinoma adjacent to gallbladder 8 by 4 by 4 cm.	Resection of neoplasm.	Lived 5 months. Rapid recurrences & widespread metastases.
6. C. T. 14 yr. girl. Malignant primary hepatoma right lobe.	Resection of neoplasm of right lobe measuring 11 by 7 by 5.5 cm.	Living & well 1 yr. 9 mos.
7. T. B. 64 yr. man. Primary carcinoma liver involving entire right lobe.	Resection of right lobe for tumor measuring 25 by 19 by 13 cm. & weighing 3500 Gm.	Died 4 hrs. after operation of shock.
8. L. B. 50 yr. woman. Primary carcinoma right lobe of liver.	Resection of a third of right lobe for excision of neoplasm measuring 14 by 11 by 10 cm. & weighing 620 Gm.	Living & well 2 yrs. 5 mos.
9. Robb. 58 yr. woman. Primary carcinoma of intra hepatic bile ducts —right lobe.	Radical resection of a third of right lobe of liver for fist size tumor.	Died during operation of cardiac arrest.

Patient	*Operation*	*Result*
10. M. S. 42 yr. woman. Cystadenocarcinoma of intra hepatic bile ducts —right lobe.	Resection of cystic carcinoma from right lobe which also was adherent to pylorus—mass measured about 15 cm. in diameter.	Lived 6 mos. Reoperated upon for recurrence—died of shock 6 hrs. after operation.
C. *Secondary Involvement of Liver by Direct Spread*		
11. N. S. 60 yr. man. Carcinoma stomach adherent to left lobe of liver.	Radical gastrectomy en masse with resection of entire left lobe of liver.	Known to have lived over 15 yrs. after operation. Lost to follow-up.
12. E. S. 64 yr. man. Carcinoma of stomach with adhesions to left lobe of liver.	Radical gastrectomy and contiguous excision of portion of left lobe of liver.	Living & well 5 yrs. 10 mos.
13. Wei. 51 yr. woman. Carcinoma of pylorus with direct extension onto upper portion of head of pancreas & onto liver at site of previous gallbladder bed (had previous cholecystectomy).	Radical gastrectomy and resection en masse of slice of upper portion head of pancreas, first portion of duodenum and portion of liver to include gallbladder bed.	Lived 9 mos. Died of carcinomatosis.
14. Z. 54 yr. woman. Carcinoma hepatic flexure with adhesion to anterior margin of right lobe of liver.	Right colectomy with contiguous resection of segment of right lobe of liver measuring 12 by 6 by 6 cm.	Known to have lived over 9 yrs., then lost to follow-up.
15, 16, 17, 18, 19 Five patients with recurrent carcinoma of stomach after previous radical gastrectomy and adhesion of recurrent mass to left lobe of liver.	Resection of gastric stump en masse with all or major portion of left lobe of liver and portions of body of pancreas, spleen and sometimes segments of transverse colon.	Survived 6 to 9 mos. Died of recurrences.
20. H. G. 39 yr. woman. Recurrent carcinoma of splenic flexure adherent to left lobe of liver.	En masse resection of left transverse and upper descending colon, portion of stomach, body of pancreas, spleen, portion of left lobe of liver, segment of jejunum.	Lived 7 mos. Died of metastases.
D. *Solitary Hepatic Metastasis Where Primary Growth has been Previously Removed*		
21. M. S. 53 yr. man. Primary rectal carcinoma resected 6 yrs. previously.	Total left hepatic lobectomy for metastasis 15 by 10 by 8 cm.	Lived 1 yr. 8 mos. Died of carcinomatosis.

D. *Solitary Hepatic Metastasis Where Primary Growth has been Previously Removed—Continued*

Patient	*Operation*	*Result*
22. L. C. 46 yr. woman. Ovarian cancer removed 1½ yrs. previously.	Resection of hepatic metastasis from right lobe measuring 14 by 10 by 7.5 cm., together with retroperitoneal metastases.	Lived 2 mos. Died of rapidly spreading metastases.
23. C. G. 60 yr. woman. Four years previously had carcinoma of sigmoid resected. Three yrs. previously total cystectomy for carcinoma of bladder.	Left hepatic lobectomy for solitary metastasis 12 by 8 by 5 cm. Reoperated upon 1 yr. 6 mos. later, partial gastrectomy for active duodenal ulcer. Both lobes of liver riddled by metastases.	Living & fairly well but has recurrences 1 yr. 11 mos. after initial left hepatectomy.

E. *Multiple Hepatic Metastases Where Primary Growth has been Previously Removed*

24. R. E. 44 yr. woman. Radical mastectomy 3 yrs. previously.	Left hepatic lobectomy for large metastasis and several smaller ones. Excision of discrete metastases from right lobe. Left hepatic lobe measured 16 by 7.5 by 3 cm. Largest metastasis from right lobe measured 4.2 by 2 by 2 cm.	Lived 1 yr. 8 mos. Died of carcinomatosis.
25. J. W. 41 yr. man. Resection of carcinoma of colon 5 yrs. previously.	Left hepatic lobectomy for metastasis 12 by 9 by 6 cm. Small metastasis resected from right lobe.	Lived 9 mos. Died of carcinomatosis.
26. A. G. 67 yr. man. Ten years previously had radical gastrectomy for carcinoma. One year previously had resection of segment of jejunum for obstructing metastasis	Total left hepatic lobectomy for large metastasis 10 by 8 by 7 cm. Five discrete metastases were resected from right lobe.	Living, well 1 yr. 1 mo. Gained 20 lbs.
27. M. N. 42 yr. woman. Panhysterectomy for leiomyosarcoma 8 months previously.	Total left hepatic lobectomy for large metastasis in left lobe and excision of metastases in right lobe. Excision of retroperitoneal metastases.	Died of shock during operation.
28. S. G. 71 yr. woman. Resection of carcinoma of colon 3 yrs. greviously.	Resection of solitary metastasis right lobe of liver, retroperitoneal metastases, vagina metastases and metastasis in lower sacrum.	Living & at work 9 months after operation. Recurrence in vagina. Liver not palpable.

F. *Solitary Metastasis in Presence of Primary Growth in Abdomen*

Patient	*Operation*	*Result*
29. H. K. 69 yr. man. Carcinoma of stomach with hepatic metastases.	Radical gastrectomy and excision of 2 discrete metastases from right lobe.	Lived 1 yr. 6 mos. Died of carcinomatosis.
30. Sol. 56 yr. man. Carcinoma body of pancreas with solitary metastasis in right lobe of liver.	Resection of body of pancreas and spleen. Excision of solitary metastasis 3 cm. in diameter, right lobe of liver.	Lived 5 mos. Died of carcinomatosis.
31. B. 72 yr. man. Carcinoma of cecum with solitary hepatic metastasis, left lobe.	Right colectomy with contiguous resection of abdominal wall in right lower quadrant. Excision of solitary hepatic metastasis left lobe, 6 cm. in diameter.	Postoperative survival satisfactory. Died of coronary occlusion 7 mos. after operation.
32. Plen. 62 yr. man. Carcinoma of pelvic colon. Solitary hepatic metastasis.	Resection and end to end anastomosis of colon with excision of small hepatic metastasis right lobe of liver.	Lived 1 yr. 1 mo. Died of carcinomatosis.
33. D. 45 yr. man. Carcinoma head of pancreas. Solitary metastasis left lobe of liver.	Pancreatoduodenectomy and excision of solitary metastasis in left lobe of liver, 6 cm. in diameter.	Returned to work. Suddenly developed rapidly progressing thrombosis inferior vena cava & died 5 mos. after operation.
34. Gr. 60 yr. woman. Carcinoma sigmoid. Metastases to many retroperitoneal nodes and liver.	Resection of carcinoma of colon with end to end anastomosis; resection of ovary; excision of solitary hepatic metastasis.	Died 5½ months later of carcinomatosis.
35. H. G. 56 yr. man. Carcinoma cecum and solitary metastasis dome of right lobe of liver.	Right hemicolectomy and excision of metastasis dome of right lobe of liver 8 cm. in diameter.	Gained 30 lbs. during survival period. Died 1 yr. 6 mos. of carcinomatosis.
36. Kolt. 51 yr. woman. Carcinoma pelvic colon.	Combined abdomino-perineal resection and pelvic lymph node dissection for carcinoma of colon. Excision of large solitary hepatic metastasis in right lobe of liver.	Succumbed at end of operation from shock.

G. *Multiple Hepatic Metastases in Presence of Primary Growth Elsewhere*

37. Bohr. 39 yr. man. Carcinoma descending colon. Two retroperitoneal node metastases. Left lobe of liver riddled with small metastases. One small metastasis dome of right lobe.	Resection colon carcinoma with end to end anastomosis. Left hepatic lobectomy—the lobe and metastasis measured 18 by 11 by 8.5 cm. Small metastasis excised from right lobe.	Lived 7 mos. Died of carcinomatosis.

G. Multiple Hepatic Metastases in Presence of Primary Growth Elsewhere—Continued

Patient	*Operation*	*Result*
38. Serg. 56 yr. woman. Multiple large masses in liver.	Excision of multiple large metastases from right lobe of liver. Aggregate weight: 594 Gm. Primary growth not discovered. Histologic diagnosis anaplastic carcinoma.	Lived 3 mos. Died of carcinomatosis.

H. Hepatic Metastases in Presence of Recurrent Carcinoma Elsewhere in the Abdomen

39. Greenb. 54 yr. woman. Two years previously had excision of carcinoma of the cecum.	En masse excision of abdominal wall in right lower quadrant, recurrent carcinoma of cecum and adherent 45 cm. of small bowel and 15 cm. of ascending and transverse colon. Resection of wedge of right lobe of liver 12.5 by 6 by 1 cm. containing a metastasis 5 cm. in diameter.	Lived 9 mos. Died of carcinomatosis.
40. Dalt. 69 yr. man. Carcinoma pylorus with multiple hepatic metastases.	Partial gastrectomy, spontaneous rupture of hemorrhage from multiple hepatic metastases forced their excision to stop bleeding.	Died 2nd postop. day. Cause? Coronary occlusion?
41. J. Gr. 55 yr. woman. Resection of carcinoma of ascending colon two years previously.	Resection of recurrent carcinoma of ascending colon en masse with right lower quadrant of abdominal wall. Excision of large (9 cm.) metastasis in right lobe of liver (upper portion).	Death at end of operation from cardiac arrest which failed to respond to adequate treatment (cardiac massage, etc.)

Summary of Operations upon the Liver

Total right hepatic lobectomy	3
Total left hepatic lobectomy	9
Large resections of right or left hepatic lobe (at least 9 cm. in diameter)	13
Multiple hepatic metastases excised	2
Solitary hepatic metastases, small tumor (less than 4 cm. in diameter) or limited resection of liver	14
Total	41
Operative mortality	6 (15%)

The factor of cardiac arrest during the operation was of importance in this series. Two of the six operative deaths were due to this cause and once it occurred, treatment proved ineffectual. The treatment consisted of thoracotomy, cardiac

massage, injection of adrenalin, and calcium chloride. These accidents occurred early in the writer's experience in radical hepatic surgery and in reviewing the events in these cases it was apparent that in each instance it was a question of a large metastasis in the upper portions of the liver. The compression and rotation of the liver that obtained in the excision well might have constricted completely the inferior vena cava just below the heart, causing the latter to become partially emptied of blood. More recent experience in heart surgery has shown how slight rotation of the heart can alter the physiology of conduction in it. With all attention focused upon the operation and the control of hemorrhage it seemed as though attention to the arrested heart was delayed for too long a period and a revival was not possible. With the appreciation of this possibility and the newer knowledge of anesthesia under these circumstances the dangers of such possibilities should be reduced.

In the other 4 fatalities, death ensued 24 hours after operation in 1 patient (case 41) as a result of marked debility of the patient. The abdomen had been opened and a carcinoma of the pylorus with large hepatic metastases was found. The latter fissured spontaneously resulting in severe hemorrhage and they had to be enucleated and their sites packed in order to prevent immediate fatal hemorrhage. In 2 other instances (cases 27 and 36) fatal shock from hemorrhage occurred. In the fourth case, a debilitated alcoholic, death ensued four hours after termination of a complete right hepatic lobectomy presmuably from shock, but necropsy was not obtained.

On the credit side of the picture there are the following satisfactory results:

One patient with enormous infiltrating angioma necessitating resection of practically the entire right lobe of the liver and portions of the infiltrated abdominal wall, the tumor 25 cm. in greatest diameter, is living and well over three years.

One patient with probably angiosarcoma who had complete right lobectomy is living two years and four months from initial operation and four months after second operation for small recurrences.

Two patients with primary carcinoma of the liver are living and free from evidence of disease one year nine months and two years five months respectively after major resections of the right lobe.

One patient who had the entire left lobe of the liver resected of a large metastasis and five smaller metastases resected from right lobe, and who had radical gastrectomy for carcinoma 10 years previously, is living and free from evidence of active disease one year and one month following excision of metastases.

One patient who had radical gastrectomy for carcinoma of stomach adherent to left lobe of the liver and had the latter entirely resected en masse with stomach is known to have lived over fifteen years but is lost to follow-up.

One patient with carcinoma of hepatic flexure adherent to right hepatic lobe and had large segment of this resected en masse with right colon is known to have lived over nine years and is lost to follow-up.

One patient had had radical gastrectomy for carcinoma that was adherent to left lobe of liver and had adherent hepatic segment resected en masse with stomach is living and well 5 years 10 months.

In some other instances of metastases palliation was achieved even though survival did not exceed 18 months.

The rationale for the attack upon large solitary metastases is obvious but insufficient evidence is available to settle the question finally as to whether such procedures are worth-while.

In animals, immunity to tumor growth has been induced by permitting a tumor to grow, removing it and then testing for regrowth of tumor. In the experience reported above, immunity of the liver to subsequent growths of metastatic tumors in it has not yet become apparent but it is a problem to be further explored, as instances of palliation have been observed.

SUMMARY

Primary hepatic neoplasms may be resected with reasonable safety. Various types of partial hepatectomy are described. Total right or total left hepatic lobectomy is quite feasible.

Adherence of a primary growth in the stomach or colon to the liver does not contraindicate en masse resection of the primary growth and a portion of the liver. Survival for five or more years is possible after such operations.

The excision of single or multiple hepatic metastases has certain theoretic justifications but as yet limited experience prevents definite impressions concerning the value of such procedures although palliation has been observed in some instances.

ADDENDUM

Since the above report three additional total right hepatic lobectomies have been performed, one of which was an operative mortality.

REFERENCES

1. Brunschwig, A.: Surgery of hepatic neoplasma, with special reference to secondary neoplasms, Cancer *6:* 725 (July) 1953.
2. Huard, P., and Meyer-May, J.: Les abces du foie, Paris, Masson & Cie, 1936.
3. Lortat-Jacob, J. L., and Robert, H. G.: Hepatectomie droite, reglee, Presse med. *60:* 549 (April 16) 1952.
4. Romieu, C., and Brunschwig, A.: Bacteriologic study of human liver, Surgery *30:* 621 (Oct.) 1951.

THE SURGICAL TREATMENT OF GASTROJEJUNOCOLIC FISTULA

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In 1899 Braun[2] first reported and discussed gastrojejunal ulceration. Since that time much has been written about the etiology, course and therapy of such ulceration and also the possible complications, notably, bleeding, perforation, severe pain and gastrojejunocolic fistula.

Before elective gastric surgery became popular, fistula formation usually was associated with malignant change in the stomach or large intestine, but as gastrojejunostomy came to be accepted in the treatment of peptic ulcer, this surgical procedure became the most common cause of fistula formation. This presentation is concerned with cases of this type.

INCIDENCE

In 1935, Lahey and Swinton[4] analyzed 1,089 collected cases of gastrojejunal ulcer diagnosed within the preceding seven years. The incidence of ulcer formation following gastrojejunostomy alone varied from 1.7 per cent to 24 per cent and following gastrojejunostomy with subtotal gastrectomy from 0.4 per cent to 10 per cent. The consensus of opinion is that fistula formation is a complication of gastrojejunal ulcer in approximately 10 per cent of all cases in which medical advice is sought. In a series of cases recently reported by Lowdon[6] in which 46 gastrojejunocolic fistulas occurred in 41 patients, the time interval between the initial surgery and the onset of symptoms varied from 1 month to 21 years (fig. 1). Since much gastric surgery has been done within the last 20 years, it is important that the surgeon keep these findings in mind. Obviously, he must make an effort to avoid this complication in the operating room and to think of it in following the patient subjected to surgical therapy for ulcer.

SYMPTOMS

The symptoms of gastrojejunocolic fistula strongly suggest the diagnosis (fig. 2). Loss of weight, diarrhea, weakness and fatigability usually are present. Fecal vomiting without evidence of obstruction sometimes is present and, if so, is pathognomonic of fistula. Nausea, fetid breath and anorexia are common, and hematemesis with melena may occur. When the condition is chronic, protein deficiencies, lowered prothrombin levels, hypochromic anemia, dehydration and metabolite deficiencies indicate the starvation present. Rarely, a patient is encountered in whom there are no symptoms suggesting the diagnosis.

DIAGNOSIS

The diagnosis is confirmed by careful roentgenographic study. Nearly always the fistulous tract may be demonstrated by means of a barium enema, while a

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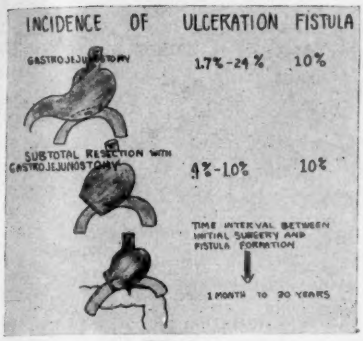

FIG. 1

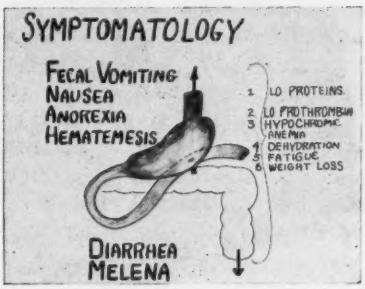

FIG. 2

FIG. 1. Incidence of ulceration and fistula occurrence following gastrojejunostomy
FIG. 2. Symptoms of gastrojejunocolic fistula

barium meal only rarely reveals the presence of a fistula (fig. 3). As many of the patients have symptoms compatible with the diagnosis of gastrojejunal ulceration only, both examinations should be made in a complete study when a patient experiences postoperative difficulties.

TREATMENT

Since gastrojejunocolic fistula first was recognized, the treatment has been surgical. Wilkie,[9] in 1934, deploring the high mortality rate of single stage operations, first directed attention to the advantages of a two stage procedure (fig. 4). In 1939, Pfeiffer[7] reported the mortality rate for one stage procedures as ranging from 25 per cent to 63 per cent in cases collected from the literature. He presented a multiple stage procedure successfully done in 3 cases, in 1 of which the surgeon was Dr. Ralph Colp (fig. 5). The first stage consisted of a proximal loop colostomy. Thereafter, all 3 patients gained weight and became symptom-free; also, the metabolite derangement was corrected. At the second procedure, done when the condition of the patient warranted, the reaction at the site of the fistulous tract had subsided markedly, and technically the procedure of excising the

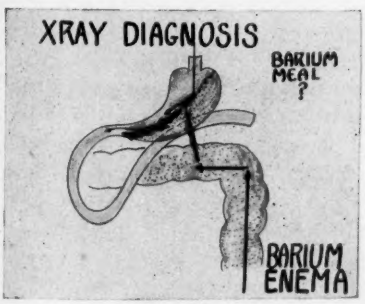

FIG. 3

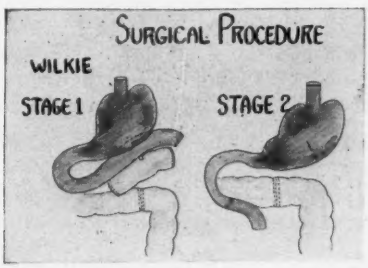

FIG. 4

FIG. 3. Schematic representation of roentgenographic diagnosis
FIG. 4. Illustration of Wilkie procedure

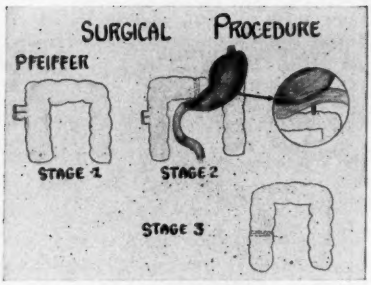

FIG. 5. Illustration of Pfeiffer procedure

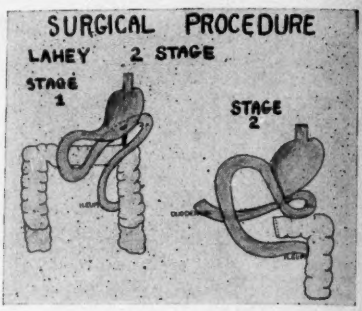

FIG. 6. Illustration of Lahey procedure

fistulous tract and re-establishing the intestinal continuity was less difficult. The proximal vent was closed in a third procedure when the patient had fully recovered from the second operation.

Lahey,[7] in a discussion of the paper by Pfeiffer,[7] described a two stage procedure which he had used successfully in 2 patients (fig. 6). In the first procedure he produced a proximal vent by doing an end to side anastomosis between the distal portion of the ileum and the descending colon. In both cases, following this bypassing operation, the patient improved as in the cases of Pfeiffer.[7] The second stage procedure consisted of removal of the cecum, ascending colon, transverse colon, involved segment of the jejunum and distal portion of the stomach, with re-establishment of intestinal continuity by an antecolic gastrojejunostomy of the Hofmeister type following an end to end suture of the jejunum. These two procedures utilized the same principles; they produced a proximal vent which apparently placed the fistula at rest and allowed normal food handling by the stomach and the small intestine.

The success of the proximal vent or bypass and the demonstrated ability of the barium enema to reveal the fistulous tract when the barium meal revealed no communication, focused attention upon the regurgitation of colonic content into

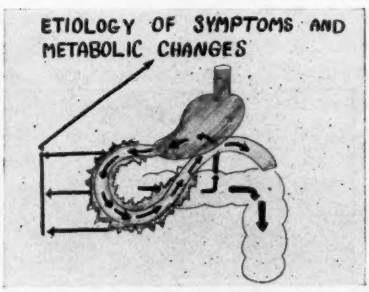

FIG. 7

RESULT

REPAIR	CASES	GOOD	POOR
	115		
SIMPLE RESTORATION	33	42 %	58 %
FISTULA CORRECTION PLUS SUBTOTAL GASTRECTOMY	75	96 %	4 %
GASTRO ENTEROSTOMY	6	68 %	32 %
GASTRO ENTEROSTOMY PLUS VAGOTOMY	1	100 %	

FIG. 8

FIG. 7. Schematic representation of circus movement

FIG. 8. Results of surgical treatment of gastrojejunocolic fistula as reported by Localio, S. A.; Stone, P., and Hinton, J. W.[5]

the stomach—first mentioned by Bolton and Trotter[1] in 1920—as an explanation of the intestinal dysfunction (fig. 7). Roentgenographic demonstration of a circus movement of barium from the stomach to the small intestine, thence to the large intestine, on to the stomach and so again into the small intestine strengthened this explanation. In 1946, Renshaw, Templeton and Kiskaddon[8] presented convincing evidence of this fact in their report of observations upon 2 dogs with gastrojejunocolic fistula and 4 dogs with gastrocolic fistula. In all of these animals, symptoms and metabolite changes comparable to those occurring in man developed in from two to six months following surgical creation of the fistula. From their studies these authors concluded that sufficient aliment passes from the stomach into the upper portion of the small intestine to maintain adequate nutrition, that passage into the colon through the fistulous tract is negligible, that deranged digestive and absorptive functions of the small intestine produce the syndrome, and that this derangement is the result of the passage of colonic contents through the small intestine.

Upon the basis of the observations and conclusions cited, the treatment of gastrojejunocolic fistula resolves itself into two problems: (1) elimination of the fistula with re-establishment of intestinal continuity, and (2) correction of the ulcer diathesis. Localio, Stone and Hinton[5] stressed the importance of elimination of the ulcer diathesis by stating that there was simple restoration of intestinal continuity following correction of the fistula in 33 of 115 cases collected from the literature of the preceding 10 years (fig. 8). Of this group, in only 14, or 42 per cent, were the results classified as good. Correction of the fistula plus subtotal gastrectomy was done in 75 of these patients' cases, with good results in 72, or 96 per cent. Gastroenterostomy alone was done in 6 patients, with good results in 4, or 66 per cent, and gastroenterostomy plus vagotomy in 1 with good results. These authors did not state their criteria of classification, but the importance of dealing with the ulcer diathesis is reflected in their evaluations. With the wide acceptance of vagotomy as the treatment of choice for gastrojejunal ulceration following gastric resection, it is noteworthy that Byrd[3] reported 2 cases in which fistula developed without pain as a symptom after vagotomy had been done for relief of gastrojejunal ulcer.

CASES CITED

My interest in this subject was aroused by 2 cases which came under my personal observation. In both patients the initial operation was a postcolic gastrojejunostomy. One case was reported by Byrd[3] in his discussion of fistula following vagotomy for gastrojejunal ulcer. In his article there is no discussion of the operative treatment. A one stage surgical procedure was done without incident on Feb. 3, 1949. The subsequent course was complicated by jaundice of progressive severity, and the patient died on the seventh postoperative day. At autopsy, the operative area was intact, and it was established that death was caused by hepatic failure secondary to advanced biliary cirrhosis.

In the second case the patient was operated upon at Riverside Hospital in 1951. There was a history of a gastrojejunostomy made elsewhere in 1943 for symptoms

compatible with peptic ulcer. The patient had not been symptom-free since the operation. For the preceding two months the symptoms had been typical of gastrojejunocolic fistula, and the pain had been of such severity as to lead to morphine addiction. Following roentgenographic visualization of the communication, a three stage procedure as described by Pfeiffer, with a subtotal gastric resection at the time of fistula excision, was done over a six month interval with successful eradication of the fistula and disappearance of all symptoms.

DISCUSSION

One cannot afford to be dogmatic about the ideal treatment of gastrojejunocolic fistula for each method of attack has advantages and disadvantages. With a proximal colostomy the right colon is fixed, there is contamination of the wound area, and closure of the proximal vent adds a third procedure to which the patient is subjected. Nevertheless, when facilities are limited, and in patients who are extremely ill, the advantages of this type of procedure far outweigh the disadvantages. The Lahey procedure avoids the third stage, and with it there is no contamination of the operative field, but there are two surgical procedures, the first of which is of major proportions when compared with a proximal colostomy. The single stage procedure is being done successfully, but intensive fluid, metabolite and whole blood replacement is required. Also, the intestine must be adequately prepared by suitable medication preoperatively, and local edema and reaction render the technical procedure more difficult. There is a type of case for which each method is best suited, and the wise surgeon will fit the treatment to the case.

The surgical correction of gastrojejunocolic fistula is obviously difficult. The utilization of all possible precautions in the surgery of peptic ulcer therefore becomes mandatory. Especially important in prophylaxis are: adequate gastric resection, proper placement of postcolic anastomosis or utilization of an antecolic anastomosis, short loop gastrojejunostomy, and aseptic surgery with complete hemostasis. Utilization of these known principles may greatly lower the incidence of gastrojejunal ulcer and of gastrojejunocolic fistula.

SUMMARY

The incidence, symptoms and diagnosis of gastrojejunocolic fistula following gastric surgery are briefly reviewed, and the evolution of the surgical treatment of this lesion is discussed. A plea is made for individualization of the method of surgical therapy. Prophylactic measures in gastric surgery are stressed as a means of reducing the incidence of gastrojejunocolic fistula. The salient features of 2 illustrative cases are presented.

REFERENCES

1. Bolton, C., and Trotter, W.: Clinical observations on jejuno-colic fistula following gastro-jejunostomy, Brit. M. J. *1:* 757, June 5, 1920.
2. Braun: Verhandl. d. deutsch. Gesellsch. f. Chir., *28:* Part 2, 94, 1899; cited by Pfeiffer, D. B.[7]

3. Byrd, B. F., Jr.: Gastrojejunocolic fistula following vagotomy for marginal ulcer, Gastroenterology *17:* 431 (March) 1951.
4. Lahey, F. H., and Swinton, N. W.: Gastrojejunal ulcer and gastrojejunocolic fistula, Surg., Gynec. & Obst. *61:* 599 (Nov.) 1935.
5. Localio, S. A.; Stone, P., and Hinton, J. W.: Gastrojejunocolic fistula, Surg., Gynec. & Obst. *96:* 455 (April) 1953.
6. Lowdon, A. G. R.: Gastrojejunocolic fistula, Brit. J. Surg. *41:* 8 (Sept.) 1953.
7. Pfeiffer, D. B.: Value of preliminary colostomy in correction of gastrojejunocolic fistula, Ann. Surg., *110:* 659 (Oct.) 1939.
8. Renshaw, R. J. F., Templeton, F. E., and Kiskaddon, R. M.: Gastrocolic fistula; clinical and experimental study, Gastroenterology *7:* 511 (Nov.) 1946.
9. Wilkie, D. P. D.: Jejunal ulcer: some observations on its complications and their treatment, Ann. Surg. *99:* 401 (March) 1934.

SPECIFIC BLOOD TRANSFUSIONS

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Those of us who have been either ordering or giving blood transfusions over the past 25 years appreciate the advances made in the technic of blood transfusion, in the equipment available for use and most of all we appreciate the advances made in grouping, typing and cross matching of the blood. However, with the increased availability of blood donors, with the ready availability of blood in our hospitals and with the speeches that have been given on the increased safety of blood transfusions, we often are prone to order blood without realizing the tremendous responsibility which the blood transfusion team must feel. Having helped prepare and administer blood for transfusions for the past 26 years, the lessons learned bring me to the following conclusions: First; that any patient who is sick enough to require a blood transfusion is too sick to withstand any type of reaction and, second; I deplore seeing a pint of blood given just to be "*doing something*" for the patient. It is my opinion that when a pint of blood is ordered, it should be as a specific point of therapy and the blood should be selected just as carefully as any other type of medication. It is on this latter point which I wish to dwell today.

Let us review briefly the methods of giving blood transfusions in 1954. True we are all using disposable equipment. The commercially available types of transfusion equipment include the vacuum glass bottle containing a solution of ACD, the plastic bag containing ACD, the gas-filled plastic bag and the plastic bag with its connected ion-resin exchanger.

Let us now examine these various types of equipment and decide in which cases we should use them. The glass bottle containiug ACD solution under vacuum with its sodium citrate content removes the ionized calcium and therefore prevents coagulation of the blood. The unionized calcium remains in solution and is found with the blood in the bottle. This is the standard bottle of blood that we find on the shelf in almost every blood bank over the Nation. It is of tremendous value. It is used to replace the loss of blood during or following surgery, or as a result of trauma. It is used in preoperative patients to elevate the hemoglobin and red blood count. It is used to elevate the blood count in the treatment of various anemias. It is not efficacious in elevating the white count or the platelet count. The platelets are almost all destroyed within a matter of a few hours in a glass bottle because of its *wetable* surfaces. This is especially true when it is drawn under vacuum. The white blood cells probably are not efficacious for over 90 minutes in this type of transfusion equipment. We therefore believe that the glass bottle which is used routinely is most efficacious in replacing blood loss or in "*building up*" a patient prior to surgery.

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Now let us separate the types of transfusions given in glass bottles; first, red blood cell transfusions. These have a definite place in a rapid preoperative preparation of a patient, where we wish to raise the red blood count and hemoglobin over a short period of time. The administration of red blood cells reduces almost by 50 per cent the volume transfused and therefore, doubles the efficacy of the transfused volume. It can also be used to great advantage for the rapid "*building-up*" of a patient with anemia. Each 500 cc. of transfused red cells will raise the peripheral blood count approximately one million in contrast to the half million elevation that we expect following the transfusion of a pint of whole blood. The next type of transfusion from the glass bottle is that of liquid plasma. This also has specific uses and there are places where nothing else will take its place. In blood loss which is not severe, plasma may be used. It should be used when whole blood is not available, even if the blood loss is severe. If we need circulatory volume and osmotic pressure, but do not need the hemoglobin to carry oxygen, plasma is just as efficacious as whole blood. It is of tremendous value in either primary or secondary shock, and if shock is due to hemorrhage of course we should add blood as rapidly as it is available. We avoid the use of plasma in cases of poisoning of the red cells, such as by carbon monoxide, where we specifically wish the oxygen carrying capacity. Liquid plasma is also efficacious in the treatment of hemophilia. The "*old fashion homemade plasma*" stored at room temperature seems to work as well for this as any other type. One word might be said about the use of serum albumin. It is used in place of plasma where we want a salt poor fluid which will elevate the plasma osmotic pressure. Since it is not a blood product that is prepared in the average blood bank, we will say no more concerning this product.

The use of the plastic bag containing ACD solution is the next to be considered. In this method blood is drawn by gravity, thereby eliminating the possibility of rupture of red cells when the blood is placed in the container. The donor set is an integral part of the container and the needle on the set is coated. The advantage of this is very obvious, for it helps prevent the coagulation of blood because the blood comes in contact with only *nonwetable* surfaces and second, it prevents the disintegration of blood platelets. We believe that its use is of value for transfusion in cases of thrombocytopenia, especially without bleeding. We believe it should be used where there is a decreased platelet count, in patients with purpura and in patients with poor clot retraction.

The gas-filled plastic bag is used by replacing the nitrogen with 50 cc. of 1 per cent sequestrene sodium in 0.7 per cent sodium chloride. We now have a container without citrate which is sometimes very desirable, especially if numerous transfusions are to be given and the patient has thrombocytopenia. Sequestrene has a very low toxicity in the quantities used. It saves even more platelets and white blood cells than does ACD solution and we use it especially where we want more thromboplastinogenase which is the enzyme present in blood platelets. In using sequestrene, the calcium from the blood replaces the two sodium molecules in the radical, forming sequestrate which is found in the blood, but has no further effect and is eliminated from the body. This type of equipment probably saves

more platelets for actual transfusion to the patient than any type available at the present time, the only exception being the concentration of platelets for transfusion. We use this type of bag and solution in purpura of all types and in those patients where transfusions are continued over a long period and the physician wishes to avoid an excess of citrate. It may be used alternately with the type next described.

The next type is the plastic bag with the ion-resin exchanger. This has a place which can be substituted by no other piece of equipment. By virtue of the fact fact that the calcium unites with the resin in the exchanger and forms an insoluble compound which remains attached to the beads, the blood not only retains its thromboplastinogen, but in addition we have added no anticoagulant to the blood. We have, in essence, whole blood which has been decalcified. When this blood is returned to the blood stream, the relatively high calcium content in the blood stream is more than adequate to recalcify the transfused blood and gives us the same effect as we would derive from direct blood transfusions. However, the use of the ion-resin exchanger removes over 60 per cent of the platelets from the blood and this must be considered. We use this type of transfusion in hemophilia where thromboplastinogen is the component of the clotting mechanism which is absent or decreased. This deficiency can readily be determined by use of the prothrombin consumption test described by Quick. It also may be used if one suspects a deficiency of factor 5 (or the labile factor) which again can be tested for in the chemistry laboratory. We also have used this type of transfusion in cases of circulating anticoagulants which either occurred spontaneously or following transfusion. We also use this type of transfusion as an alternate method where very large volumes of blood are to be given.

The next type of transfusion has been forgotten by many in *"the rush for the new"*. It is the direct blood transfusion. This is a procedure which many have never used, especially those who have grown up with transfusions in the last decade. There still is a place for the direct blood transfusion, and this place has not been filled by any of the other methods which we presently have at our command. It is of value in cases of afibrinogenemia, for example, as a result of abruptio placenta or severe acute liver damage. It is also of tremendous value in cases of prolonged postoperative bleeding, where the bleeding may be due to a great extent to vascular fatigue. It is also of value in cases of severe oozing in which other types of transfusion have failed to give relief from the apparent bleeding tendency. It is also of value in cases of hemophilia, where again we want thromboplastinogen and we do not want the citrate or any other anticoagulant. It is also of value in those cases where a circulating anticoagulant either can be demonstrated or where, because of the nature of the bleeding, one is quite assured that a circulating anticoagulant is present. I make a plea for the continued use of this staunch ally. Those of us who have used it over the years and who have not abandoned or forgotten it, find that we deplete our blood bank reserves less often by resorting to direct transfusion in more instances.

A few words might be said about two other products: one is antihemophiliac globulin which also is known as globulin X. This product was designed specifically

for the treatment of hemophiliacs who were either bleeding or were being prepared for surgery. It has been used and abused in many other types of cases. We must remember that this antihemophiliac globulin is antigenic and its indiscriminate use may produce, on subsequent injection, circulating anticoagulants with resultant hemorrhages much more serious than those which we were originally trying to control. Another product which we must remember is fibrinogen which is now commercially available and is specific for the therapy of afibrinogenemia.

There is one type of bleeding in which we do not suggest transfusions except where the blood loss endangers the life of the patient. I refer to hereditary familial purpura simplex which is a condition occurring spontaneously in females who develop bruises and ecchymosis with minimal trauma and have a normal platelet count. These individuals are not helped by transfusions except for replacement when the deficiency requires such treatment.

CONCLUSIONS

In closing, I might say that while we can transfuse and we can continue to obtain blood, we must search for the cause of the bleeding by careful study of our patients and then select the type of blood we believe is "*tailor-made*" for this particular patient at this particular time. We must add to this the other types of therapy, both medical and surgical, which have been demonstrated to be efficacious in the control of blood loss, so that the use of transfusions may be limited to the number really needed, thereby reducing the opportunity for possible reactions.

THE MANAGEMENT OF INTRAEPITHELIAL CARCINOMA OF THE CERVIX

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There is renewed interest in the lesion *intraepithelial carcinoma*. In part this is due to the development of exfoliative cytology study and its evident value in detecting early suspicious lesions. Since this lesion has been described in the vulva, vagina, cervix uteri and uterus, breast, prostate, skin and other areas, it is necessary that the clinician, pathologist and cytologist agree on definition. The original description by Rubin[7] is clear and hardly could be improved upon. It is as follows:

"1. An indistinct uncertain definition of cell outline, particularly in the deeper layers (germinal, proliferating).

2. The presence of irregular, large, intensely stained nuclei occasionally grouped in clumps.

3. No definite stratification, only partial parallelism of the basal cells (more often they are seen to be irregularly disposed, or at a slant toward the tunica propria).

4. The marked nuclear granulation. This sign deserves special attention because, according to Schottlaender . . . it is very frequently seen in carcinoma which has not yet undergone cornification."

Actually in a clear, well-cut, well-stained section one can fairly readily identify such an area. The clinician is perhaps too demanding in asking, is this *malignant* or *non-malignant*. The pathologist oftentimes gets involved in the criteria of malignancy, invasion, point of origin, epidermitization, infection, *preinvasion* and other points. The cytologist is on surer ground when he speaks in terms of *atypicalities*. If anything else were needed to add mild confusion, after explaining *basal cells* and *reserve cells* it is to learn that the patient is pregnant.

In an attempt to anticipate, diagnose and treat patients with intraepithelial carcinoma of the cervix it is well to recall the early work of Rubin,[7] Cullen,[2] Meyer,[4] Novak,[5] and more recently TeLinde,[10] Hertig and Younge,[12] Pund,[6] Carter,[1] Erickson,[9] and many others. The excellently conceived and documented monograph of Stoddard's[8] has complete references, carefully studied patients and a hope for future information.

DIAGNOSIS

Our[3] evaluation of the aid from exfoliative cytology studies has been recorded. In the diagnosis of uterine cancer by studies of genital smears we are constantly

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concerned with the identification of intraepithelial carcinoma of the cervix. We have been interested in this problem because of its importance to the patient, because of similar lesions of the cervix which occur during pregnancy, and of the possible advantages the method may have in the differential diagnosis.

Certain benign conditions engender nuclei in cervical epithelium which may be confused with the atypical nuclei of the intraepithelial lesion. However, through the cooperation of a conservative Department of Pathology we have been able to recognize cellular elements that are sometimes associated with intraepithelial carcinoma of the cervix.

The patients in the cases reported were examined in the years 1947 to 1949, inclusive. Smears from 10,029 patients were studied. In 44 patients (0.44 per cent) the pathologic diagnosis was found to be intraepithelial carcinoma of the cervix.

Invasive carcinoma was not ruled out in 7 patients. The genital smears in 4 of them were interpreted as squamous cell carcinoma of the cervix. In 1 of these 4 patients the clinical impression was carcinoma. There was a clinical impression of squamous cell carcinoma of the cervix in 4 of the 44 patients. Ten patients presented no evidence of a pathologic condition. On examination 26 patients had what were thought to be benign conditions of the cervix.

Three-fourths (75 per cent) of the lesions occurred in patients under 45 years of age. Twenty-one (47.7 per cent) of these patients were between 35 and 45 years of age.

The smears in 11 patients (25 per cent) did not show elements suggestive of intraepithelial carcinoma of the cervix. Smears in 2 of these patients were unsatisfactory because of poor fixation and insufficient epithelial constituents. Twenty lesions (45 per cent) were suspected. Thirteen (29.5 per cent) were not differentiated cytologically from invasive carcinoma. Twenty-seven (61.4 per cent) of the lesions were found in 1949. Seventeen of them were detected in the last six months of the year.

It is apparent that the majority of patients who have intraepithelial carcinoma of the cervix did not show clinical signs or symptoms of the lesion.

Experience is required in identifying intraepithelial carcinoma of the cervix. In interpreting this lesion from smears, the cytologist inaugurates procedures which may entail serious economic or social problems for the patient. This responsibility and that of detecting the lesion in surveys make us more conscious of the fact that investigators in this field should be exceedingly well trained, and more important, that these cytologists should work in close cooperation with conservative pathologists.

The pathologic diagnosis of intraepithelial carcinoma of the cervix in pregnant patients presents a problem. We have been interested in the possibility of distinguishing, in smear preparations, the lesion which may regress during the puerperium from the one which will persist as carcinoma in situ.

Patients with intraepithelial carcinoma of the cervix comprised 0.44 per cent of 10,029 patients who were checked in the Department of Obstetrics and Gynecology during the years 1947, 1948 and 1949.

Clinically, 4 of these patients were thought to have carcinoma of the cervix, whereas 26 patients were thought to have only benign cervical lesions. The cervices of 10 patients appeared normal.

Thirty-three of the 44 patients with intraepithelial carcinoma of the cervix were under 45 years of age.

Six patients were pregnant.

Thirty-eight patients (86.4 per cent) were white.

Again it should be clear that the diagnosis is made by tissue study. We prefer a knife biopsy that goes up to the internal os. If necessary, the entire endocervix is removed for study.

Carter's[1] report from Duke, in which we participated, has two remarkable charts, a questionnaire which he sent to members of the American Gynecological Society and the American Association of Obstetricians, Gynecologists and Abdominal Surgeons and a related one circulated by Davis to the same group. Carter posed the question regarding treatment in age groups (from 20 to 62) and parity (from 0 to 4 living children) none of whom showed invasion. There were 6 categories in these hypothetic patients. His summary was:

"The results of our questionnaire are interesting. The use of hysterectomy rises with the age of the patient. There is also a commendable tendency in the first 4 patients (aged 20, 26, 32 and 40 years) to preserve the ovaries.

Cauterization would be used by only 8 in the treatment of the first 4 patients and would not be used by anyone in the last 2 patients.

Radium and x-ray therapy would be used by one of our members for all 6 patients. Three would use x-ray and radium in Patient 3; 10 in Patient 4; 12 in Patient 5; and 14 in Patient 6 (the older age group).

Radium alone would be used by one member in Patient 1; by 2 in Patient 2; by 2 in Patient 3; by 4 in Patient 4; by 2 in Patient 5; and by 3 in Patient 6.

No one suggested the use of radical operation for Patients 1 and 2. One would use x-ray and radium and radical operation with node dissection in Patient 3; 3 would use the radical operation with node dissection in Patients 4, 5, and 6.

In Patients 1, 2 and 3 cervical amputation would be used by 10, 9 and 4 members, respectively; in Patients 4, 5 and 6 by 1, 1 and 1 respectively."

Davis worded his question a bit differently and was more concerned with classification, regression, cytology and *adequate treatment* and his summary was:

"1. The majority classified cancer in situ as Stage 0 cervical cancer and considered it as the preinvasive stage of true cervical cancer.

2. The majority did not believe that spontaneous regression of cancer in situ occurred and did not believe that it regressed during or after a pregnancy. The majority also did not accept a tissue diagnosis of cancer in situ during pregnancy.

3. The majority had never in their own personal experience observed spontaneous regression of cancer in situ. Fifteen stated they had observed it in 1 or 2 patients; 6 had observed it in 3 patients and 3 stated that they had observed "probable" regression.

4. The majority could not eliminate the possibility that regression following adequate biopsy of the cervix may not have been due to the excision in toto of the lesion.

5. Only a small majority used routine cytologic techniques prior to biopsy.

6. The majority favored multiple punch biopsy or cold-knife cone biopsy and but 2 used the cutting current for biopsy specimens.

7. The vast majority favored continued study when repeat positive cytologic reports and negative pathologic reports were returned.

8. For adequate treatment of cancer in situ the majority favored total hysterectomy; but 10 used electroconization and coagulation; 6 used cervical amputation; 4 preferred radical operation; 4 favored irradiation and radical operation; 2 employed irradiation alone; 1 stated that when total hysterectomy was done the vaginal hysterectomy was preferred. Seventeen stated categorically that radical operations were not indicated. Fifty-three stated that the type would vary with the age of the patient.

9. Seventy believed that cancer in situ should be followed by frequent repeated cytologic studies and biopsies with operation withheld. Seventy-six did not agree to delaying the operation.

10. One hundred thirty-four had never found lymph node involvement, or had never heard of lymph node involvement, when careful studies of the cervical tissue by the pathologist showed only cancer in situ. Eight had heard of lymph node involvement."

TREATMENT

The treatment should be influenced by several factors. From the available data we can know that the patient with carcinoma in situ is likely to be a decade younger than the patient who has fundal malignancy. We should remember the incidence of suggestive changes, both cytologic and pathologic, found in pregnancy. Some of these lesions undoubtedly have been removed in their entirety by an adequate biopsy specimen operation. None can deny the possibility of regression in some of the lesions, especially in pregnancy. The lesion is most often found in an unsuspected cervix when routine cytology or biopsy is done. The subjective symptoms are not remarkable; perhaps postcoital bleeding is the most likely. The age, parity, and wishes of the patient should be considered; the patient must give permission for operation. We have followed a patient who first had this suspicious lesion at age 18, and two months in her first pregnancy, through two pregnancies and who at present has negative findings. Another patient in her late twenties has literally had her cervix removed by repeated biopsy examinations and who now shows no atypicalities of cells. In our opinion, expecially in the younger age group, we do not have the compulsion to institute *total* operations that is found in early (Stage I) invasive malignancy of the cervix. Contrarily, one should not forget the surgical homily, "the early diagnosis and complete removal is the only known cure for cancer." But is this cancer? In all likelihood there is a *preinvasive* stage before invasion. The literature is filled with this discussion. Few have had the temerity to follow *intraepithelial carcinoma* over a period of years to see if it did develop invasion, a histologic picture that all will agree is malignant. This, in our opinion, can and should be done. These patients can be followed by exfoliative cytology studies and biopsy examination. Our active treatment is influenced by the above enumerated qualifying factors.

We have not employed irradiation or radium, although we can see where their use might be necessary and judicious. Surgery is the usual treatment and complete hysterectomy, going wider parametrially and excising more of the vagina is our choice of operations. In a patient 45 years old or more it is our practice to include bilateral salpingo-oophorectomy. Naturally, we get permission to include any procedure in any age group, including radical nodal dissection, when visual or palpatory evidence warrants further procedure in the interest of a complete operation and the eventual welfare of the patient.

REFERENCES

1. Carter, B., et al: Methods of management of carcinoma in situ of cervix, Am. J. Obst. & Gynec. *64:* 833 (Oct.) 1952.
2. Cullen, T. S.: Cancer of the Uterus, Philadelphia, W. B. Saunders Co., 1909.
3. Kaufmann, L. A., Cuyler, W. K., and Ross, R. A.: Intraepithelial carcinoma of cervix, Surg., Gynec. & Obst. *91:* 179 (Aug.) 1950.
4. Meyer, R.: Die epithelentwicklung der cervix und portio vaginalis uteri und die pseudoerosio congenita (congenitales histologisches ektropium) Arch. f. Gynak. *91:* 579, 1910.
5. Novak, E. L.: Pathologic diagnosis of early cervical and corporeal cancer with special reference to differentiation from pseudomalignant inflammatory lesions, Am. J. Obst. and Gynec. *18:* 449 (Oct.) 1929.
6. Pund, E. R., and Auerbach, S. H.: Preinvasive carcinoma of cervix uteri, J. A. M. A. *131:* 960 (July 20) 1946.
7. Rubin, I. C.: Pathological diagnosis of incipient carcinoma of uterus, A. J. Obst. *62:* 668, 1910.
8. Stoddard, L. D.: Problem of carcinoma in situ with reference to human cervix uteri: reprint, chap. 8, Progress in Fundamental Medicine, Philadelphia, Lea and Febiger, 1952.
9. Stoddard, L. D., Erickson, C. C., and Howard, H. L.: Further studies on histogenesis of intra-epithelial carcinoma and early invasive carcinoma of cervix uteri, Am. J. Path. *26:* 679 (April) 1950. Abstract.
10. TeLinde, R. W.: Cancer-like lesions of uterine cervix, J. A. M. A. *101:* 1211 (Oct. 14) 1933.
11. Younge, P. A.: Preinvasive carcinoma of cervix, Arch. Path. *27:* 804 (April) 1939.
12. Younge, P. A., Hertig, A. T., and Armstrong, D.: Study of 135 cases of carcinoma in situ of cervix at Free Hospital for Women, Am. J. Obst. & Gynec. *58:* 867 (Nov.) 1949.

SOME COMPLICATIONS OF HEAD INJURIES

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The vast majority of the literature on head injuries deals with the acute phase or the immediate effects of trauma. Skull fractures, studies of force, its transmission and effects; concussion, contusion and laceration of the brain; and edema and intracranial hemorrhage, are the common subjects of discussion. The acute phase of head injuries is varied, spectacular, engaging and demands more action. It rightfully has occupied most of our attention in an effort to save life and ward off or reduce to a minimum the complications and disabling sequelae of head injuries.

Disabling sequelae of head injuries may occur either as a direct result of trauma, or as a result of complications that follow trauma. In either case, some complications and sequelae cause varying types of physical disability or mental impairment that may become a real challenge and problem to the local physician or surgeon in his attempt to improve or rehabilitate his patient. Foremost among the questions one must ask himself in such cases are,—has a cerebral atrophy occurred, is there a hydrocephalus, a concealed or quiescent blood clot within the head, some vascular lesion, or a spontaneous accumulation of air intracranially.

Many times the cause for the continued impairment of function, mental changes or disability is not apparent on examination. It is only understood when the actual cerebral changes are revealed by pneumography, or other special neurologic studies.

It is for these reasons that I wish to recall for you some complications of head injuries with their disabling sequelae that you may encounter months or even years after injury. I hope to point out the organic basis for the disability and in some cases what may be done to help or rehabilitate the patient.

ACUTE TRAUMATIC INTERNAL HYDROCEPHALUS

Sometimes, early, as the result of trauma to the head, an acute internal hydrocephalus may develop. One or both ventricles may dilate to varying degrees. The cause of this condition has not be determined.[6] A blockage of the cerebrospinal fluid at the foramen of Monroe, third ventricle, the aqueduct or the incisura of the tentorium, because of a blood clot, swelling or adhesions conceivably may cause a ventricular dilatation. Also externally, the subarachnoid pathways may not permit the normal movement of the fluid outward to the pacchionian bodies, or the pacchionian bodies may be less able to dissipate the fluid because of pressure, adhesions and red blood cells.

Clinically this condition may simulate the signs and symptoms of acute sub-

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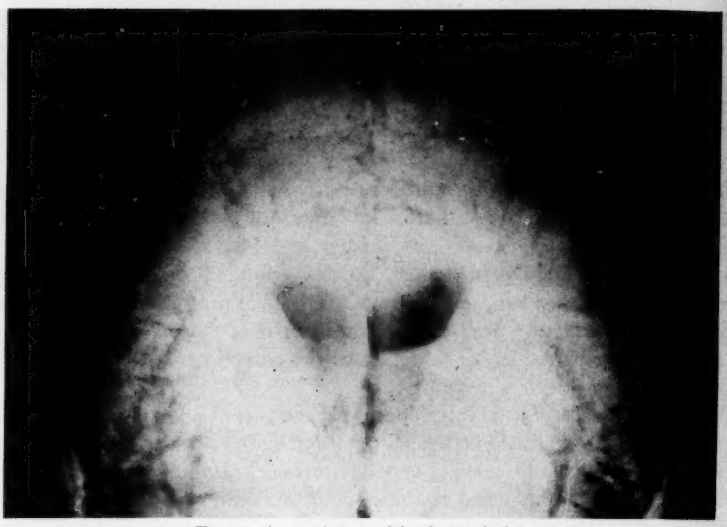


FIG. 1. Acute internal hydrocephalus

dural hematoma. The patient is usually unconscious, or nearly so, with some focal signs, as pupillary changes, facial weakness of supranuclear type, paresis of one side of the body and reflex changes. The acute manifestation may linger on, and it becomes evident that some complication has occurred.

Although air studies are most often withheld until the subacute or chronic state, however, when employed the dilated ventricles can be found soon after injury (fig. 1). Pneumography can be used to good advantage in these cases. A diagnosis is established early. Therapy and a prognosis can be predicated on what is found, thus eliminating a long period of observation and expectant treatment.

TRAUMATIC CEREBRAL ATROPHY

Traumatic atrophy of the brain may result from direct injury to the tissue, indirectly as the result of disturbed physiologic processes, such as anoxia and swelling, and because of vascular injury. It may be local or diffuse (figs. 2 and 3).

At the time of the initial injury a successful reduction of intracranial pressure, maintenance of a good air way, oxygen and good supportive measures may be effective in eliminating some of the cerebral damage and consequently some of the later symptoms.

After the acute symptoms have subsided, patients with gross cerebral damage still complain of headache, dizziness and simply of not feeling well. Varying degrees of mental impairment with personality changes may be noticed. There may be some focal signs, such as spasticity of the extremities or a motor involvement of the arms or legs. The patient is unable to return to his former employment or perhaps now is doing some easier and simpler job. He may have seizures.

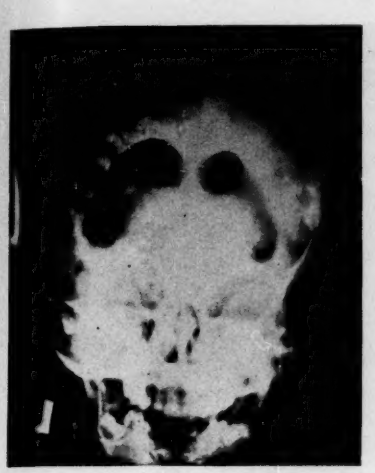


FIG. 2

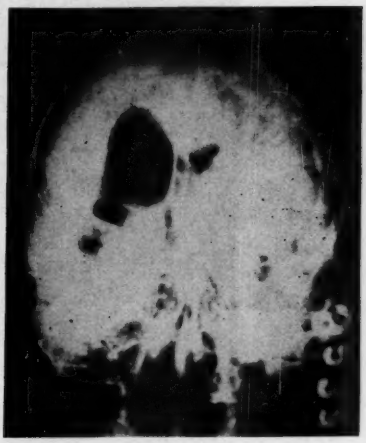


FIG. 3

FIG. 2. Cerebral atrophy following trauma
FIG. 3. Unilateral cerebral atrophy following trauma

Encephalography with a determination of the status of the intracranial structures will help in making a prognosis and determining the correct plan of treatment. Also it may be a source of considerable relief to the family and to the patient himself to know the exact reason for his various unexplained group of symptoms and his inability to carry on as he had before injury.

INTERNAL CAROTID—CAVERNOUS SINUS FISTULA WITH PROPTOSIS

A traumatic fistula between the internal carotid artery and the cavernous sinus may produce an ipsilateral proptosis, a bruit over the temporal region and along the course of the artery. Vision may be greatly impaired by virtue of the anterior displacement of the eye and the lack of motion of the globe. The lids are usually edematous and will not cover the cornea (fig. 4). Ulceration of the cornea may follow. The patient usually complains of a headache and dizziness, also the constant audible bruit is very annoying. He is disabled and uncomfortable.

This lesion does not always develop immediately after injury. Its appearance may be delayed for a considerable time. The trauma may be severe or perhaps even termed slight. At times the communication between the artery and the cavernous sinus may close spontaneously with or without compression of the carotid artery in the neck. If not, surgery is employed.

The lesion is amenable to surgery, but the patient must be prepared preoperatively for at least two weeks, sometimes longer, during which time he is taught to compress the common carotid artery with his thumb for periods of 10 minutes or longer while lying in bed. This is repeated many times a day. During this time it is possible to evaluate the procedure by noting whether the eyeball recedes, the venous congestion is diminished or the bruit is relieved. Also the

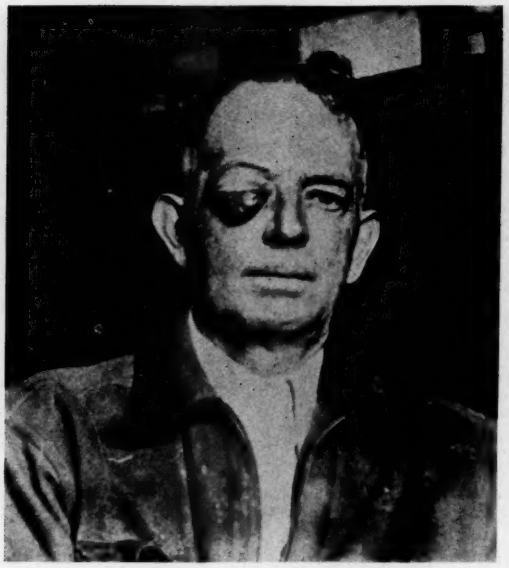


FIG. 4. Proptosis. Internal carotid cavernous sinus fistula

general effect of the carotid occlusion should be studied closely for motor weakness or changes of consciousness.

Usually a collateral circulation is developed so that sooner or later the common carotid artery may be ligated. The usual precautions are taken for releasing the artery immediately if untoward signs are noted. However, an adequate preparation will reduce the likelihood of trouble. This procedure usually gives very gratifying relief of all symptoms and returns the patient to a normal state.

CHRONIC SUBDURAL HEMATOMA

The chronic subdural hematoma may lay relatively dormant and without noticeable signs for weeks or months. Symptoms may develop so long after trauma that the injury, which is usually trivial, may have been forgotten. The insidious nature of the lesion makes it quite difficult to apprehend, so that many changes may take place before the clot is removed, and not a few escape attention and remain unrecognized until found at necropsy (tables I and II).

In only 4 per cent of our cases was the trauma sufficient to produce a fracture of the skull. It is usually trauma, such as bumping the head against a door, or against some object when bending over. Sometimes there may be a more violent injury, such as a car accident, or a fall, but seldom is consciousness lost. So the patient goes along with his usual duties until symptoms gradually develop.

The subjective symptoms occur first.[2] Headache that gradually increases over a period of weeks, perhaps is the first complaint. Home remedies are used in ever increasing amount, but to no avail. Dizziness and vertigo occur intermit-

TABLE I

Incidence of major symptoms

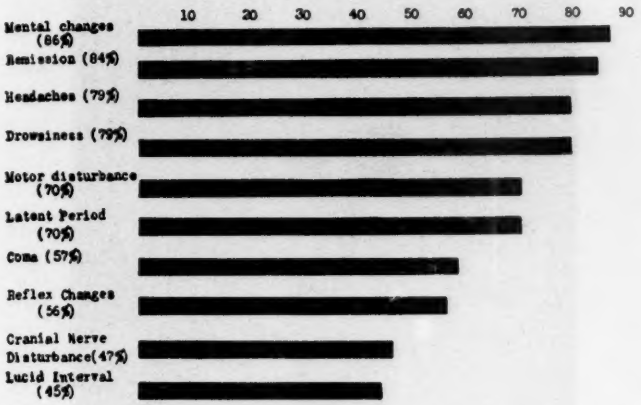
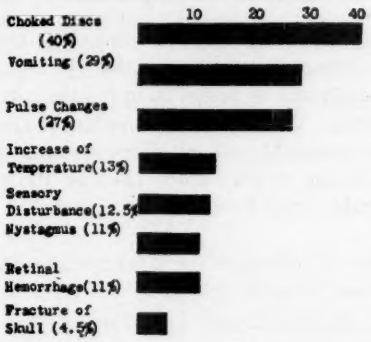


TABLE II

Incidence of minor symptoms



tently. Personality changes and impaired integrated processes become quite evident and eventually become prominent. At this point a good neurologic examination may reveal some blurring of the discs, some pupillary changes, motor deficit, reflex changes or visual field defects. If the pineal is calcified, roentgenograms will show a shift to the opposite side. This is an important and very helpful observation. It can serve to lateralize the clot. However, both sides, if possible, should be explored at operation.

The treatment is removal of the hematoma. Operation usually can be done under local anaesthesia which is preferable. Patients who have cerebral trauma do not tolerate barbiturates especially pentothal. Ether may be used. If the condition of the patient will permit, a bone flap will give the best exposure for removal of the subdural clot. This clot is organized to a greater or less degree.

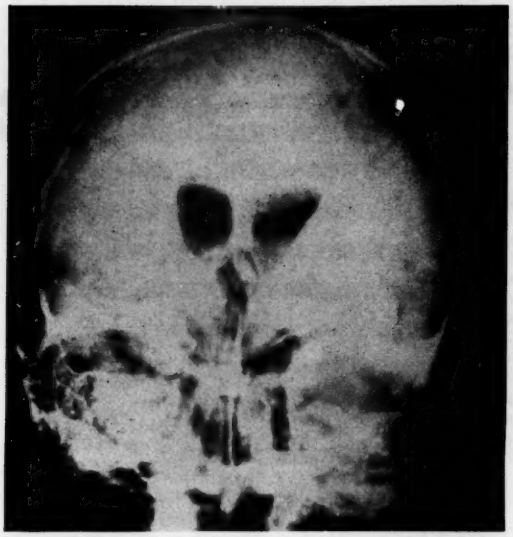


FIG. 5. Cerebral atrophy. Sequela of subdural hematoma

However, if the condition of the patient is unsatisfactory for a long operative procedure, or if it has been determined that the clot is liquid or semi-liquid, a triangle burr hole arrangement is made. A burr hole is made in the temporal, frontal and parietal areas. The burr hole is enlarged as needed. The dura is opened and the clot is removed with pituitary spoons, suction and lavage. A through and through lavage may be made between the burr holes. The brain then is distended by introducing fluid into the ventricle or the spinal subarachnoid space.

Since this lesion may lie dormant for a long while, the brain immediately beneath may suffer from pressure and from the irritating effect of bilirubin, which is formed from the hemoglobin by action of the mesothelial cells that rapidly form a membrane about the clot. As a result, thrombosis of the underlying vessels, compression of the brain, with organic changes of a permanent type, may occur. Unilateral cerebral atrophy (fig. 5) with associated contralateral motor changes, or perhaps mental impairment, remain as sequelae after the clot is removed.

AEROCELES AND RHINORRHEA

A cranial aerocele is a spontaneous collection of gas beneath the pericranium or within the skull. (For details concerning this lesion, see recent paper by the author.[5]) It occurs most often as a complication of skull fractures. Incomplete repair at the site of injury, mild infection and a dural tear, may tend to produce a fistulous tract between one of the cranial accessory sinuses and the intracranial cavity. This, accompanied by a cerebral laceration, may produce the most common type of aerocele, a pneumocephalus.

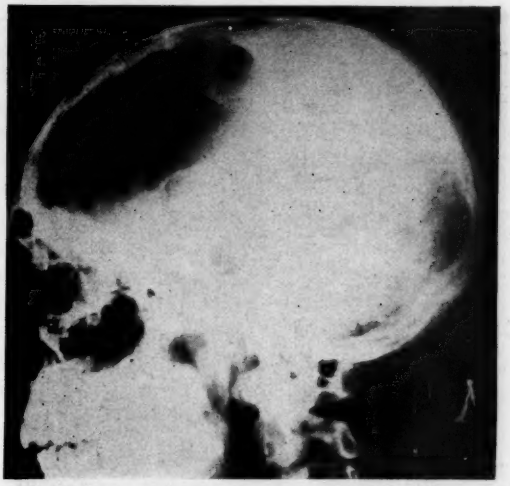


FIG. 6. Right frontal aerocele communicating with ventricle

Its development is delayed in the majority of patients, weeks and even months after injury. They are not suspected and seldom diagnosed until found by roentgenogram. By this time, the quantity of air may be sufficient to cause destructive changes in the cerebrum, especially if it accumulates within the brain as a pneumocephalus. In such a case, focal signs from the area involved, as well as signs of increased intracranial pressure will be present.

The following case report will tend to illustrate the symptomatology and treatment.

CASE REPORT

G. A., a white male, aged 17, was injured in a car accident and received emergency treatment at a hospital near the site of injury. A laceration above the right eye was sutured. When seen 48 hours later, there was considerable swelling in the right frontal area and about the eye. He was stuporous, but rational. There were no focal signs. Roentgenograms showed a frontal fracture into the right frontal sinus. He recovered normally, the wound healed readily and he was discharged on the seventh day.

Six weeks later he reported back to the office. During the last two weeks headaches gradually became more severe and he showed some emotional imbalance, with definite euphoria. There were no objective signs. Unfortunately, we did not suspect his trouble. Roentgenograms were not retaken. He returned six weeks later in a slight irrational state with marked personality changes. Headache, nausea and vomiting had progressed. Roentgenograms showed a right frontal pneumocephalus (figs. 6 and 7).

On Sept. 25, 1948 a bone flap was turned down in the right frontal area. The convolutional markings were ironed out, and the brain was distended. On palpation it was easily compressible, as if there were a cavity filled with gas beneath. The thin distended cortex collapsed when the cavity was opened, and the gas escaped. The cavity held approximately 8 oz. of fluid when filled. The interior of the cavity was smooth and clean. We could see that it extended almost to the very tip of the frontal lobe. It was noted that there was a communication between it and the frontal sinus. Ten thousand units of penicillin were introduced into the fluid-filled cavity. Extradural exposure was then made back to the point

FIG. 7. Antereoposterior view of right frontal aerocele

where the fistulous tract communicated between the frontal sinus and the pneumocyst. This was severed with Bovie and a piece of gelfoam saturated with penicillin was placed in the opening over the distal end of the fistulous tract. A transplant of pericranium about 3 by 3 cm. was sutured over the fistulous opening in the dura. The brain and the dura were not separated. The dura was sutured and the remainder of the closure was made as usual. He was given antibiotics. Roentgenograms made on October 1, showed the bone flap in the right frontal region, and only a slight quantity of air could be visualized in the cystic cavity. On Nov. 3, 1948 all the air had been absorbed.

This patient has been observed carefully. He has presented some signs of euphoria, and there still remains definite personality changes. Generalized myoclonic seizures have occurred on two occasions. He, however, has been able to control the seizures entirely in the past year with anticonvulsive drugs. He has since been married, and has been working steadily.

A perceptible spinal fluid rhinorrhea may or may not accompany a pneumocephalus. Either may be separate and independent, but usually they occur together. The fluid is replaced by air. At times the patient is not clinically able to discern the dripping of fluid from the nostrils or ears or the quantity may be so meager that it is not noticed. The same mechanism and cause produce both of the lesions.

In some basal fractures, after the swelling has subsided and the clot retracted, a spinal fluid leak will appear. It may occur even months after injury.[1, 3] The flow may be intermittent if a ball-valve mechanism is operating, or if stored temporarily in the sphenoid sinus, which empties when the patient leans forward. A continuous flow would indicate a large patent opening.

Approximately 4 per cent of skull fractures are sooner or later accompanied by a spinal fluid leak from the nose or ears. Meningitis or intracranial abscess formation is to be feared.[2, 7]

Antibiotics, maintenance of the same degree of elevation of the head, and expectant therapy otherwise for five days to a week is justified at first to see if the fluid leak will stop spontaneously. This frequently occurs, but if it does not occur surgical repair must be carried out.

In open injuries of the head, a proper and thorough closure of the dura and repair of the bony defect at the time of injury, will eliminate most of the aeroceles and rhinorrhea. Closed injuries, however, with fracture into the sinuses or ear require a direct attack upon the fistulous tract between the intracranial structures and the ear or sinuses, with the closure of this tract and repair of the dura. Sometimes it is necessary to seal the crevices in the base of the skull with bone wax, gelfoam soaked in thrombin or other substances one may have confidence in using.[5]

CONCLUSIONS

Acute traumatic hydrocephalus is a recent observation. It may not be an entity. It simulates an acute subdural hematoma. It has been mentioned so as to stimulate an interest in it with the hope that more may be learned about the lesion as more cases are reported.

Post-traumatic atrophy of the brain may account for many subjective symptoms, such as mental and personality changes, as well as actual focal motor signs. Encephalography will help the attending physician to understand the patient's problem. It will permit a better prognosis and treatment.

Chronic subdural hematomas require frequent mention. It is only through constant awareness of this insidious lesion that more of them may be apprehended and lives saved, because they respond well to surgery.

Aeroceles should be kept in mind when dealing with head injuries that have not responded well to treatment and especially if a history of a rhinorrhea is given.

It is hoped that some of the complications here mentioned may come to mind when confronted with a patient with prolonged post-traumatic symptoms and disability. They may be due to a hydrocephalus, a cerebral atrophy, a concealed blood clot within the head, an arteriovenous fistula, or a spontaneous accumulation of air intracranially.

REFERENCES

1. Adson, A. W.: Cerebrospinal rhinorrhea: surgical repair of a fistula: report of case, Proc. Staff. Meet. Mayo Clin. *16:* 384–387 (1941).
2. Dandy, W. E.: Pneumocephalus (intracranial pneumatocele or aerocele), Arch. Surg. *12:* 949 (May) 1926.
3. Gurdjian, E. S., and Webster, J. E.: Surgical management of compound depressed fracture of frontal sinus, cerebrospinal rhinorrhea and pneumocephalus, Arch. Orolaryngol. *39:* 287–306, 1944.
4. Jelsma, F.: Chronic subdural hematoma, J. Arch Surg. *21:* 128 (July) 1930.
5. Jelsma, F.: Cranial aerocele, Am. J. Surg. *87:* 437 (March) 1954.
6. Raaf, J. A.: Internal hydrocephalus, a complication of acute cerebral trauma, Am. J. Surg. *87:* 431 (March) 1954.
7. Smith, J. S., and Malcolmson, P. H.: Traumatic pneumocephalus, Canad. M. A. J. *30:* 650–651, 1934.

THE TREATMENT OF INOPERABLE PROSTATIC CARCINOMA WITH Au 198

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Cancer of the prostate is considered to be the most common malignancy in men after the age of 60 and probably occurs in 20 per cent of all men who have passed that age. In routine autopsy studies six pathologists found small latent foci of prostatic carcinomas in 14 to 46 per cent of all men over the age of 50. Ten per cent of routine benign prostatic specimens, when rechecked and intensively examined, also were found to be malignant. With the increase in life expectancy all of us have become more respectful of this malignancy of the aged, since its frequency will, no doubt, increase with longevity.

The cause of cancer of the prostate is unknown although it certainly would appear to be the result of some chemical or hormonal imbalance. It generally is agreed that 90 per cent of prostatic carcinomas originate in the posterior lobe. The lesion is slow growing and usually metastasizes late. For this reason carcinoma of the prostate is asymptomatic until extension beyond the confines of the capsule, or distant spread, has occurred.

The imperativeness of rectal examination cannot be overemphasized since digital palpation is the most valuable single diagnostic factor in detecting early cancer of the prostate. It has been estimated that one can accurately diagnose benign prostatic hypertrophy in 96 per cent of all cases and carcinoma in 88 per cent by rectal examination.

It generally is agreed that early prostatic carcinoma should be treated by radical prostatectomy. Patients between 75 and 80 years of age, whose life expectancy is only a few years, possibly should not be subjected to radical prostatectomy for they do not tolerate the procedure well at this age. It has been estimated that only 5 to 10 per cent of prostatic carcinomas are diagnosed sufficiently early to be cured by radical surgery. This small percentage is the result of (1) the failure of early prostatic carcinoma to produce symptoms, (2) the failure of the examiner to make periodic rectal examinations, and (3) the failure of male patients to voluntarily seek periodic rectal examinations.

Over 150 years ago John Hunter observed that castration produced atrophy of the normal prostate gland and in the latter part of the nineteenth century White reported that castration was of benefit in 85 per cent of the patients with obstructive prostatism. However, it has been only in the past 15 years that endocrine therapy for the prostatic cancer has been introduced and developed.

The first reports of estrogenic management of prostatic carcinoma were made by Huggins[6] and others in 1941. At about this time Munger[8] used external radiation to decrease the androgen factors in the treatment of prostatic malig-

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nancy. In 1945 Herbst and Sauer[5] introduced radiation of the pituitary gland and Huggins and Scott[7] observed the effect on prostatic carcinoma by removing the adrenal and pituitary glands.

One of the most important factors to consider in inoperable carcinoma of the prostate is the patient's age. Certainly life expectancy is greater today than ever before and as a result we are seeing more men with advanced cancer of the prostate. Insurance charts show that life expectancy for a 75 year old man is six years. Since the life span has been increased we are striving to affort the increasing number of patients afflicted with prostatic malignancy longer and more comfortable survival periods.

Actually, however, the survival time has been increased very slightly. Nesbit and Plumb[9] in 1946 reported that in 795 untreated prostatic carcinomas the average survival from time of diagnosis was 21.2 months. A series of 485 cases presented by Bumpus[1] showed a survival time of 31 months. These patients were also untreated by estrogenic therapy. Survival at the present time, by utilization of all forms of palliative therapy, has increased only an additional 9 to 18 months.[4] This makes us wonder whether this is a real or an apparent increase in survival time, since it would appear logical to assume that we, undoubtedly, are seeing prostatic carcinomas earlier today than a few years ago. It is entirely possible that the increase in survival time may be in direct proportion to the increased interest in geriatrics and improvement in general care of the aged.

To realize that estrogenic therapy, despite its widely heralded virtues, really has contributed so little to the increase in survival time of the patient with inoperable cancer of the prostate, is, to say the least, most surprising and, at the same time, disappointing. It is no doubt a realization of these facts that prompted Flocks[2] in 1951 first to instill a radioactive isotope, Au 198, into the prostate gland of patients with hopeless carcinomas of that organ. Since his original report Flocks now has reported the cases of more than 200 patients treated by this method. His results have been sufficiently encouraging to stimulate other investigators along the same lines of endeavor.

It was determined years ago that irradiation with the roentgen ray or radium was ineffective in the treatment of cancer of the prostate. The question which immediately arises is why would the use of a radioisotope be more effective than roentgen therapy or radium.

Isotopes have several unique properties not possessed by roentgen ray, radium, radon, or even cobalt as presently employed. Most important of these is that an isotope is a form of radiation suspended in a solution as a stable colloid containing millions of small particles. These small particles represent multiple point sources of radiation which are separated by varying distances.

Au 198 was selected as the most suitable isotope for use in prostatic cancer because of its stability, its favorable half-life of 2.7 days, its relative uniformity in arrangement of its molecular pattern, its proportion of gamma and beta radiation and its relatively low cost of production.

Problem of dosage. In the use of any radioactive isotope, which is injected interstitially for the treatment of cancer, the problem is not simply one of how

many millicuries to use but rather how the radioactive material is distributed throughout the tumor and how long it remains at the site of the injection.

The cancerocidal dose in terms of roentgens delivered to the tumor has been fairly well determined in the case of most tumors by years of treatment with radium and roentgen ray and it has been shown that optimal results are obtained by using a dose which falls within fairly narrow limits. *Under*dosage results in a therapeutic failure whereas *over*dosage may actually decrease rather than increase the cure rate and produce many undesirable sequelae and complications. Although this dosage can be rather simply determined with the roentgen ray, the problem is much more complex in the case of interstitially injected radioactive isotopes. The dose received from beta and gamma radiation is easy to calculate if it is assumed that there is an absolutely uniform distribution of the isotopes throughout the tumor, providing that the exact size and volume of the tumor is known. Unfortunately, these ideal conditions never occur in actual practice. The problem then, actually, is trying to determine the varying doses in different parts of the injected tissue since it is not the average dose we are interested in but rather the maximum and the minimum doses.

In the use of radioactive gold interstitially, the problem of radiation dosage is that of gamma and not beta dosage. At first glance, this statement seems rather surprising in view of the fact that the beta radiation roughly is 10 times greater than the gamma rays, if one assumed an equal distribution throughout the tissues. Since gold does not have a homogeneous distribution the radiation must be computed from multiple point sources of radiation which are separated by varying distances. The beta particles of radioactive gold have an effective range of only 1 millimeter and any tumor cells which were not within 1 millimeter of the radioactive gold would be unaffected by this radiation. The effect would be that while some of the cells would be intensely irradiated by beta particles, other tumor cells would receive no radiation whatsoever. With gamma radiation the dose also drops off fairly rapidly from a point source due to the inverse square law. With multiple point sources there is cross firing of tumor cells from many different points of radiation with the result that all of the tissue is irradiated although there may be a difference of more than 100 per cent between the minimum and maximum dose, assuming that the point sources are not separated by a distance of more than 5 millimeters. If the distance between the point sources is greater than 5 millimeters, the difference between the minimum and maximum dose will be much greater and a point is finally reached where portions of the tumor are receiving less than a cancerocidal dose.

In order to diminish the difference between the minimum and maximum dose, it is necessary to produce as uniform a distribution of the isotope as possible in the injected tissues. Having determined the most desirable volume, how many millicuries should be used in order that the minimum dose will approach the known cancerocidal dose and the maximum dose will not be large enough to produce undesirable sequelae?

The importance of knowing these factors must be self evident since the results to be obtained in treating patients with carcinoma of the prostate will depend to

a large extent on how carefully the radiation is administered. These factors can be determined experimentally although this has not been done up to the present time. By obtaining injected material from both man and animal, it would be possible by radioautograph to determine the distribution of the gold, and the variation in dosage received by different portions of the prostate could be determined by making multiple radio assays of different portions of the prostate.

Indications and contraindications for use of Au 198. The best chance of cure of cancer of the prostate, which is confined within the capsule of the gland, is total prostatectomy including removal of the seminal vesicles. The use of radioactive isotopes in such a condition is definitely contraindicated. Evidence of spread of the cancer beyond the area of the prostate and its immediate contiguous structures, as determined by marked elevation of the serum acid phosphatase, physical examination, or bony metastases, precludes the usefulness of interstitial radiation with an isotope. The general condition of the patient and any serious complicating illnesses must also be considered before using this type of therapy.

DISCUSSION OF CASES

Our results in the treatment of 26 consecutive cases of advanced carcinoma of the prostate with Au 198 have been for the most part very encouraging (tables I and II). The earliest cases of any series are, as a rule, subject to the greatest complications and the poorest results. Conversely, as the technic is improved and selection of patients is better, the end result becomes more gratifying. We have had a total of 5 deaths and most of these occurred in the first 6 patients (table III). Likewise, almost all of the serious complications arose in the first 10 patients treated (table IV). In patients presently treated serious complications are practically nonexistent.

In describing the local changes that take place in many extensive, large, stony-hard prostates, one must exercise restraint for fear of being too enthusiastic. However, as a rule, the entire area undergoes a softening and shrinking process that results in a flat or even concave prostatic bed. Many of these patients already have had the benefit of previous estrogenic therapy and have either failed to respond to it or the carcinoma has been reactivated. Not only has an atrophy of a large percentage of viable tumor tissue taken place, but the chance of early metastasis has been minimized by the pick up of the beta particles in the lymph vessels draining the area.

The average age of the patients in our series was 68.8 years with the youngest being 52 and the oldest 81. Estimates of the size of each prostate were attempted preoperatively and the average of these estimations was 32 grams. The dose of Au 198 delivered into each gland generally was based on its size and the amount of tumor present. The average dose given per operation was 90.3 millicuries. In view of the average size this gives an average ratio of 2.1 millicuries per gram of tissue, which is higher than the reported optimum dose. The largest dose to any one patient was 200 millicuries, however, this was delivered in two separate operations at five and one-half month's interval. The largest dose given at a single operation was 145 millicuries and the smallest 25 millicuries.

TABLE 1

Case No.	Age	Serum Acid Phosphatase	Size of Prostate	Dosage of Au 198	Results
1	56	4.0	++	40 RP* 10 Pr†	Fair
2	52	3.1	+	8 RP 5 Pr	Expired
3	73	5.3	++++	112–5 RP 20 Pr	Expired
4	65	6.5	++	40 RP None	Expired
5	81	3.7	+	75 RP None	Good
6	75	6.0	++++	145 RP None	Expired
7	80	3.0	+	45 RP None	Excellent
8	67	1.4	+++	55 RP None	Excellent
9	67	4.5	+++	100 RP None	Expired
10	66	2.2	++	75 RP None	Excellent
11	78	6.5	+++	75 RP None	Fair
12	75	2.3	+++	85 RP None	Fair
13	65	3.0	+++	70 RP 10 Pr	Good
14	76	1.0	++	90 RP 10 Pr	Excellent
15	67	2.0	+++	85 RP 20 Pr 95 Pr‡	Poor
16	58	3.0	+++	90 RP 10 Pr	Excellent

* Retropubic.
† Perineum.
‡ Second admission—open perineal installation.

One of the many advantages of this type of therapy is the ease with which additional Au 198 can be injected perineally as nodules or areas of hardness develop. As many as 5 to 7 cc. of the colloidal gold may be injected through spinal needles through the perineum with no ill effects. This method has been employed on four occasions with good results.

Some pertinent remarks about individual cases may prove to be of interest:

The patient in case 1 was diagnosed in 1951 and treated with transurethral resection and orchiectomy. His acid phosphatase was 4.0 mg. but roentgenographic evidence of metastasis was lacking. In April 1953 the mass in his prostate had increased markedly with involvement of both vesicles. Au 198 was

TABLE 2

Case No.	Age	Serum Acid Phosphatase	Size of Prostate	Dosage of Au 198	Results
17	74	2.0	+++	80 RP 10 Pr	Excellent
18	75	1.5	+++	100 RP 10 Pr	Excellent
19	55	1.6	+++	95 RP 20 Pr	Excellent
20	75	2.5	++	112 RP None	Excellent
21	69	2.2	+++	100 RP 15 Pr	Poor
22	68	3.4	++	108 RP 20 Pr	Excellent
23	68	1.2	None*	80 RP 15 Pr	Excellent
24	58	4.1	++	95 RP 20 Pr	Excellent
25	70	1.8	+++	85 RP 20 Pr	Excellent
26	67	3.6	+++	105 RP 20 Pr	Excellent

* Previous prostatoseminovesiculectomy.

TABLE 3

Analysis of deaths

Case Number	Age	Remarks
No. 2	52	Died 3rd. P. O. day—massive pulmonary embolus
No. 3	73	Died 4th. P. O. day—extensive metastasis to bone and soft tissues
No. 4	65	Died 6 months P. O. of generalized carcinomatosis
No. 6	75	Died 2nd. P. O. day—extensive metastasis to liver
No. 9	67	Died 5 months P. O. of extensive metastasis

TABLE 4

Complications

Total patients involved	8
Pulmonary emboli (one recovered)	2
Delayed wound healing	5
Rectal irritation with hemorrhage	2
Postoperative bladder calculi	2
Severe postoperative nausea	2
Urethral stricture	1

injected and the result has been satisfactory. The prostatic mass had become smaller but a hard ridge on the right has recently required 2 cc. of gold injected perineally. It has not completely disappeared although the patient has never felt better.

The patient in case 5 was found to have a massive carcinoma with involvement of the left seminal vesicle in January 1953. Transurethral resection, orchiectomy and estrogenic therapy were given. In May 1953 Au 198 was injected retropubically and the mass in the prostate has shrunk to normal size. His course has been complicated by a dense urethral stricture and the development of a bladder calculus. The calculus recently was removed and progress is at present satisfactory.

The patient in case 7 was diagnosed in September 1951 and was treated with orchiectomy, transurethral resection and estrogens. The lesion was retarded until May 1953 when the area became larger. Interstitial injection of Au 198 was accomplished in May 1953 and although he has not been seen recently, he is reported to be in good health and has lost no weight.

The patient in case 8 had obvious extracapsular carcinoma of the prostate late in 1951. Transurethral resection and orchiectomy were employed and he was comfortable until May 1953 when his prostate became larger with increased hardness. He was given Au 198 retropubically in July and has had an excellent result. His convalescence was marred by a pulmonary embolus, which necessitated a caval ligation.

The patient in case 10 has received excellent results from a grade III carcinoma diagnosed first in 1949. He now has no palpable evidence of malignancy in his prostate.

The patient in case 11 has been treated for grade III carcinoma since 1949 and recently required a perineal injection of gold into a small apical mass of tumor. Otherwise he appears to be in good health.

The patient in case 12 had a large extracapsular malignancy grade II in 1949 and in March 1953 his rectum appeared to be full of tumor. He was treated with gold in August 1953 and at the present time his prostatic bed is concave except for a knot of tumor near his right apex. This will require some additional Au 198 perineally.

The patient in case 13 was diagnosed in June 1952 as having a hard fixed gland with most of the tumor in the left side of his prostate. This was injected generously in August 1953 and in October additional gold was injected into the right apex of the prostate. The original area of extensive tumor was completely free of malignancy at that time. His present condition is excellent.

The patient in case 14 has received combined therapy for a grade III lesion which was treated first in August 1953. He is in excellent health at present and has no palpable evidence of malignancy in his prostate.

The patient in case 15 was seen first in 1951 with a large and grossly irregular prostate. Transurethral resection and orchiectomy were not successful in effecting much improvement in the size of the malignancy. In October 1953 Au 198 was injected retropubically and again there was very little evidence of shrinkage.

In January 1954 the area was exposed perineally and 95 millicuries were injected. It is too early to determine results of the latest therapy.

The patient in case 16 had a large inoperable carcinoma in 1950 with involvement of the right seminal vesicle. Combined therapy has given excellent results with a flat prostatic bed. He has been distressed recently with the formation of some encrustations in the bladder neck.

The patient in case 18 has received a wonderful result from an extensive lesion involving his urethral sphincter. He was seen first in July 1953 and the combined therapy has been done.

The patient in case 19 is our youngest patient in the series and the combined therapy has resulted in remarkable shrinkage of a large hard gland. He is only four months postoperative but has no palpable evidence of a tumor.

The patient in case 20 was complicated by the finding of a large bladder tumor as well as a large prostatic malignancy. This was resected at the time of the instillation of the Au 198 and there has been no recurrence of either.

The patient in case 21 has received very little recession of his large tumor from the combined therapy. He obviously will need considerably more Au 198 perineally.

The patient in case 23 is one of the most unusual cases. He had a radical prostatoseminovesiculectomy and orchiectomy in 1946. He apparently had been followed very closely by his physician and it was only in November 1953 that symptoms of prostatism recurred. Rectal examination showed marked induration of the bladder neck and sphincter. Transurethral biopsy of a small amount of tissue revealed grade II adenocarcinoma of prostatic origin. This patient was given the largest per gram dose of the series. There was marked regression of the induration within four weeks.

Complications. Delayed wound healing and prolonged suprapubic drainage of urine has been the most frequent complication. All patients have remained hospitalized until drainage has stopped. The average period of hospitalization has been 22 days. The shortest period of hospitalization was 13 days. It is believed that the inflammatory reaction from radiation has a definite effect on wound healing but there apparently is little relationship between the millicuries/gram ratio and wound healing. There have been two pulmonary emboli. The first occurred four days postoperative and resulted in immediate death. The other occurred 27 days postoperative in a patient who was slow to heal and simply would not move or get out of bed. Recovery was uneventful in the latter case.

SUMMARY AND CONCLUSIONS

A brief outline is presented showing why Au 198 is the isotope of choice at the present time in the treatment of inoperable prostatic carcinoma. The technic of instillation and a series of 26 cases have been presented with a discussion of results obtained by retropubic and perineal instillation of Au 198.

Other subjects of great importance which have received very little attention are 1. Of the gold injected into the prostate, how much remains in the gland? How

much is lost in the urine, through drainage at the operative site, through gold spilled and removed from the body in the operating room, and gold which goes by blood stream to the liver? 2. How much gold goes to the adjacent lymph nodes and is this sufficient to have a cancerocidal effect? Flocks,[3] in a recent personal communication, is convinced that this effect is produced by Au 198. 3. What is the dose received by the rectum, adjacent to the prostate, and how can this be reduced without affecting the treatment?

These are only a few problems that arise in connection with the use of interstitial gold therapy which must be solved. By so doing we would materially improve the results of treatment as well as to eliminate, insofar as possible, undesirable radiation reactions.

REFERENCES

1. Bumpus, H. C., Jr.: Transurethral resection and paraplegic, Tr. Am. A. Genito-Urin. Surgeons (1946) *38:* 283–289, 1947.
2. Flocks, R. H., Kerr, H. D., Elkins, H. B., and Culp, D.: Treatment of carcinoma of prostate by interstitial radiation with radioactive gold (Au 198): preliminary report, J. Urol. *68:* 510 (Aug.) 1952.
3. Flocks, R. H.: Personal communication, 1954.
4. Glanton, J. B., Fitzpatrick, R. J., Orr, L. M., and Hayward, J. C.: Palliative treatment of prostatic cancer, South. M. J. *45:* 1094 (Nov.) 1952.
5. Herbst. W. P.: Effects of biochemical therapeuses in carcinoma of prostate; further observations, J. A. M. A. *127:* 57 (Jan. 13) 1945.
6. Huggins, C., Scott, W. W., and Hodges, C. V.: Studies on prostatic cancer; effects of fever, of desoxycorticosterone and of estrogen on clinical patients with metastatic carcinoma of prostate, J. Urol. *36:* 997 (Nov.) 1941.
7. Huggins, C., and Scott, W. W.: Bilateral adrenalectomy in prostatic cancer; clinical features and urinary excretion of 17-ketosteroids and estrogen, Ann. Surg *12 :* 1031 (Dec.) 1945.
8. Munger, A. D.: Experiences in treatment of carcinoma of prostate with irradiation of testicles, J. Urol. *46:* 1007 (Nov.) 1941.
9. Nesbit, R. M., and Plumb, R. T.: Prostatic carcinoma; follow-up on 795 patients treated prior to endocrine era and comparison of survival rates between these and patients treated by endocrine therapy, Surgery *20:* 263 (Aug.) 1946.

POSTERIOR VAGINAL HERNIA (ENTEROCELE)

A FREQUENTLY-OVERLOOKED CONDITION

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Although posterior vaginal hernia or enterocele has been known since Garengeot[4] described it in 1756, and although given cursory mention in most textbooks, it frequently is overlooked, probably because its possibility does not occur to the practitioner or possibly because few patients are examined while in the standing position. Rectocele is known to be a common condition; enterocele, directly above it, often passes undetected on examination or even during operation, and thereby an unsatisfactory end-result is obtained.

In 1885 Thomas[10] described a surgical attack upon this hernia. He used the abdominal approach and eliminated the sac by turning it inside out. In 1912 Moschowitz[7] described a somewhat similar procedure for cure of rectal prolapse which is equally applicable to the condition here considered. He obliterated the sac and pouch of Douglas by means of concentric purse-string sutures. In 1922 Ward[11] described a useful vaginal operation to cure the condition.

Previous to the publication of these papers, posterior vaginal hernia was described, as still frequently it is, as *high rectocele*. Other designations have been: vaginal enterocele, hernia of the pouch of Douglas, retrovaginal hernia, and rectovaginal hernia, none of which is entirely appropriate. Since there is lack of proper designation, the authors urge adoption of the term *posterior vaginal hernia*, implying that the rectovaginal pouch of peritoneum, in contrast to the vesicouterine pouch as found in anterior vaginal hernia, participates in the formation of the hernial sac.

INCIDENCE

In an analysis of 52 cases of posterior vaginal hernia, as reported by Weed and Tyrone[13] from the Charity Hospital and Ochsner Clinic, the incidence alone or in conjunction with prolapse of the uterus, cervical stump or vaginal vault, was 1 in 1000 gynecologic hospital admissions. Folger[3] found 95 posterior vaginal hernias in 52,996 admissions to the University Hospitals of Cleveland, an incidence of 0.18 per cent. A large proportion of posterior vaginal hernias follow vaginal hysterectomy for prolapsus uteri.

DEFINITION

A true posterior vaginal hernia is a saccular projection of the peritoneum through a defect in the endopelvic fascia, beginning between the uterosacral ligaments close to the posterior surface of the cervix, and dissecting its way

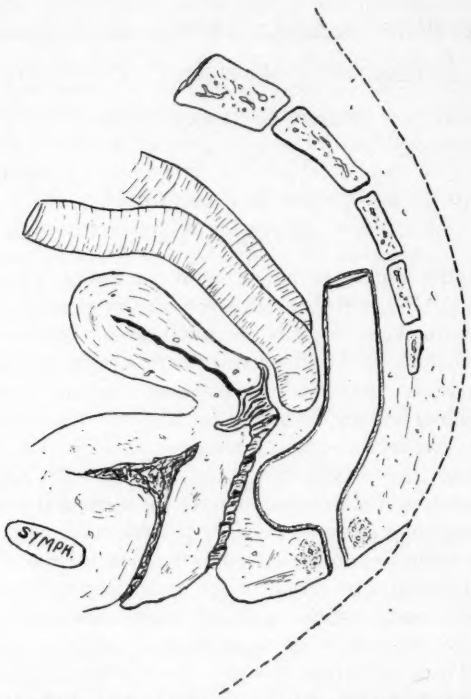

FIG. 1. Incipient true posterior vaginal hernia (congenital)

downward in the space between the posterior vaginal wall and the anterior surface of the rectum (fig. 1). In its descent along the rectovaginal septum this herniation encounters the perineal body over which it may pass, emerging as a bulging mass through the vaginal introitus. The hernial sac usually contains small intestine but occasionally adnexa, omentum, or other intra-abdominal structures. In rare instances it presents in one of the labia.

SYMPTOMATOLOGY

While the symptoms are often those associated with rectocele, there are no *specific* symptoms of posterior vaginal hernia. However, any woman who complains of bulging into the vagina, pressure in the pelvis and/or rectal difficulties should have a thorough pelvic examination to rule out its presence.

DIAGNOSIS

Suspicion is the first and essential factor in the diagnosis of the condition. One characteristic of posterior vaginal hernia is its disappearance when the recumbent position is assumed, only to recur immediately on standing or straining. Should it fail to reduce spontaneously with the patient lying down and the hips elevated, it may be easily replaced within the abdomen by slight pressure from

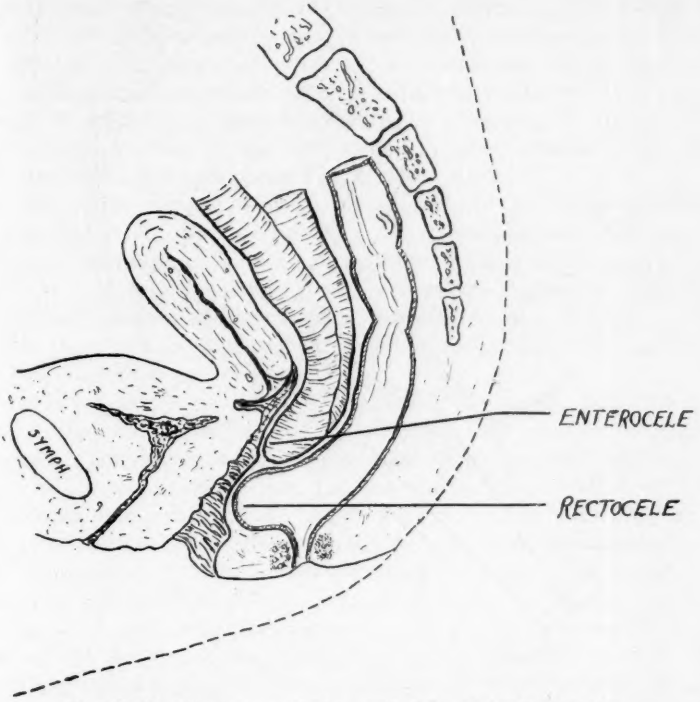

FIG. 2. Simultaneous posterior vaginal hernia and rectocele

the examining finger in the vagina. This reduction is often accompanied by a gurgling sensation as the intestines are pushed upward into the abdominal cavity.

Bueermann[1] found that stroking or pinching the hernial sac stimulates peristalsis which is plainly visible through the posterior vaginal wall. Meigs[8] stresses the importance of examining the patient in the erect position with the index finger in the rectum and the thumb in the vagina. With the patient straining, a bulge will be felt between the fingers, the abdominal contents of which frequently can be determined and the point of origin between the uterosacral ligaments and adjoining posterior cervix be ascertained. Rectocele is devoid of these features. When rectocele is present in conjunction with posterior vaginal hernia, the diagnosis is often made by inspection, as the division between the two is indicated more or less distinctly by a transverse furrow (fig. 2).

Waters[12] recommends simultaneous vaginal speculum and rectal digital examination as a means of diagnosis. After completion of the routine gynecologic examination a Graves bivalve speculum is introduced deep into the vagina and the index finger of the right hand after entering the rectum is maintained in contact with the anterior rectal wall opposite the cervix uteri throughout the

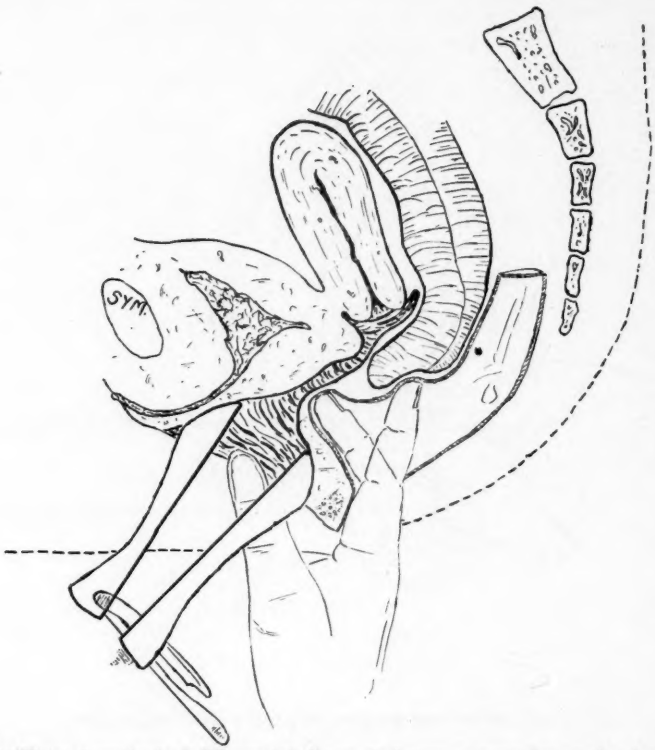

FIG. 3. Waters' method of differential diagnosis between posterior vagina hernia and rectocele.

examination. The Graves speculum is withdrawn first with the patient quiescent and then reinserted and withdrawn while the patient is straining forcibly. Should a posterior vaginal hernia be present, the bulge into the vagina begins immediately below the cervix and progressively enlarges caudad, as the speculum is withdrawn, while the anterior rectal wall is held in contact with the rectal finger. In rectocele, upon withdrawal of the speculum, the anterior rectal wall falls away from the rectal finger, since the bulge is formed by the herniation of the anterior rectal wall into the vagina. Should both conditions occur simultaneously, the two observations are noted one after the other. Figure 3 demonstrates the essential features of this diagnostic method as employed by Waters. We have found each of these diagnostic methods which is advocated by Meigs and Waters to be equally sound; they should both be used.

TREATMENT

The vaginal approach, as advocated by Ward[11] is commonly and successfully employed. The posterior vaginal hernia is opened in the midline for its entire extent and the peritoneal sac is freed by dissection up to the uterosacral liga-

ments. The sac is opened, its neck is closed by a purse-string suture placed as high as possible, the excess of peritoneum is excised, and the uterosacral ligaments are approximated with interrupted sutures. The levator ani muscles are then coapted in the midline and repair completed as for rectocele.

Should the hernia be large or complicated by adhesions, the vaginal method should be supplemented by Moschowitz's abdominal obliteration of the culdesac of Douglas by concentrically placed purse-string sutures.

Pouching of the culdesac of Douglas associated with a prolapse of the uterus is best treated by vaginal hysterectomy if it is feasible, at which time care must be taken to assure an obliteration of culdesac of Douglas and of a potential hernial space, however small it may be, between the uterosacral ligaments by their thorough approximation. Phaneuf[9] emphasizes that shortening of the vagina in these cases is an extremely important factor, for if the vagina is not shortened, recurrence is very likely.

Should the patient's age and other circumstances mitigate against the use of vaginal hysterectomy, the interposition operation may be in order. Hundley[11] has devised a simple means of disposing of the hernial sac and obliterating the culdesac of Douglas in conjunction with the interposition operation.

After invaginating the culdesac, the apex of the hernia is attached high onto the posterior surface of the uterus. It is absolutely essential to recognize and correct the posterior vaginal hernia before doing any repair for cystocele. Should the latter be repaired first, then one is plagued by a lack of exposure and, consequently, inadequate repair of the posterior vaginal hernia results. In cases associated with debility, advanced age and systemic diseases in which the above surgical approaches are not feasible, one may resort to occlusion of the vagina. The authors, as well as Phaneuf[10], have secured good results by occluding the vagina by colpocleisis, using either the subtotal method of LeFort[12] or the total method of Dujarier and Larget.[15] The operation leaves the hernia itself untouched but prevents its further development by occluding the vaginal canal.

DISCUSSION

Posterior vaginal hernia has been considered a rare condition, textbooks giving it but a few lines. Analysis of reports shows it to be more common than suspected. Alertness on the part of physicians and the awareness that posterior vaginal hernia is often mistaken for a rectocele will do a great deal toward improving diagnosis and postoperative results. We agree with the currently prevailing opinion that vaginal hysterectomy, whenever feasible, gives the most satisfactory results. By removal of the uterus, all the structures concerned are rendered more accessible to repair and can be most effectively employed to occlude the defect. As in any hernia, the more years elapsing following repair, the more recurrences will turn up. The number of cases reported by any single author over a period of years is too few to be of statistical significance.

SUMMARY

The frequency with which posterior vaginal hernia is mistaken for a rectocele is emphasized.

Differences between posterior vaginal hernia and rectocele, from the standpoints of anatomy, diagnosis and surgical correction, are defined. Useful diagnostic methods are discussed.

Posterior vaginal hernia is frequently overlooked at the time of vaginal or abdominal hysterectomy or may follow inadequate support of the vaginal vault at the time of hysterectomy.

Several methods of surgical correction, based upon the etiologic type, are described.

REFERENCES

1. Bueermann, W. H.: Vaginal enterocele; report of three cases, J. A. M. A. *99:* 1138 (Oct. 1) 1932.
2. Dujarier, C., and Larget, M.: Total colpectomy for complete genital prolpase in elderly women, J. de chir. *25:* 283 (March) 1925.
3. Folger, G. K., in discussion on Weed, J. C., and Tyrone, C. H.[13]
4. Garengeot, quoted by Bueermann, W. H.[1]
5. Hundley, J. M., Jr.: Treatment of vaginal hernia associated with prolapsus uteri, South. M. J. *20:* 345 (May) 1927.
6. Le Fort, L.: Nouveau procédé pour la guérison du prolapsus utèrin, Bull. gén. de Thérap., etc., Par. *92:* 337, 1877.
7. Moschcowitz, A. V.: Pathogenesis, anatomy and cure of prolapse of rectum, Surg., Gynec. & Obst. *15:* 7, 1912.
8. Phaneuf, L. E.: Voluminous hernia of cul-de-sac of Douglas treated by total colpectomy, Am. J. Obst. & Gynec. *34:* 152 (July) 1937.
9. Phaneuf, L. E.: Posterior vaginal enterocele; hernia of cul-de-sac of Douglas, Am. J. Obst. & Gynec. *45:* 490 (March) 1943.
10. Thomas, T. G.: Vulvar and vaginal enterocele, New York M. J. *42:* 705. 1885.
11. Ward, G. C., Jr.: Technic of repair of enterocele and rectocele, J. A. M. A. *79:* 709 (Aug. 26) 1922.
12. Waters, E. G.: Diagnostic technique for detection of enterocele, Am. J. Obst. & Gynec. *52:* 810 (Nov.) 1946.
13. Weed, J. C., and Tyrone, C. H.: Enterocele: analysis of fifty-two cases, Am. J. Obst. & Gynec. *60:* 324 (Aug.) 1950.

TANTALUM GAUZE AS A TISSUE BUILDER: HERNIA REPAIR IN TWO STAGES*

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While tantalum gauze has proved to be a most valuable material in hernia repair[2], it is bound to have certain limitations, as has every other material or method used in surgery. One case of huge incisional epigastric hernia is being reported elsewhere[3] in which the first implantation of tantalum gauze did not cure the hernia, but the implantation did cause such a proliferation of fibrous tissue through and about the meshes of the material, that abundant strong tissue was available for successful repair at a second operation. We wish to report the use of the same principle in the repair of a recurrent femoral hernia.

CASE REPOI T

Mrs. I. W., 64 years of age, white, was first seen by us in September 1949, when she was admitted to the Baltimore City Hospitals with a recurrent femoral hernia. She had a chronic nonproductive cough and an old history of tuberculosis with bilateral apical fibrotic disease. (However, cultures were subsequently negative for tubercle bacilli on multiple occasions.) At a previous admission in May 1945 she had had an initial Cooper's ligament femoral hernia repair in which No. 36 stainless steel wire was used as suture material.

Our first (the patient's second) operation was done on Sept. 27, 1949. The conjoined tendon was found to be separated from Cooper's ligament, to which it had been sutured at the first operation. It was thought best to do another Cooper's ligament repair, although the conjoined tendon was very poor. In order to facilitate bringing it down to Cooper's ligament, a relaxation incision was made in the anterior sheath of the rectus muscle.[1] The conjoined tendon was then sutured to Cooper's ligament with interrupted sutures of heavy black silk. Silk of a large caliber was used because it was thought that the failure at the first operation might have been due to the cutting through of the fine wire sutures. Due to the poor quality of the conjoined tendon, a piece of tantalum gauze 4 by 4 inches in diameter was placed over the entire area. This was sutured to Poupart's ligament below and then its center depressed so as to make contact with the conjoined tendon-Cooper's ligament suture line.[4] The aponeurosis of the external oblique was then closed over the tantalum gauze and the rest of the wound closed as usual. Silk was used throughout as suture material.

During the annual follow-up examination on this patient on Nov. 17, 1953, it was discovered that she had a recurrence, which she said had been present for almost a year. She was therefore readmitted to the Baltimore City Hospitals for the repair of her twice recurrent femoral hernia.

Our second (the patient's third) operation was done on Nov. 24, 1953. The old wound again was explored through an inguinal incision. It was found that the conjoined tendon again had pulled away from Poupart's ligament, the heavy black silk sutures having cut through it, as was evidenced by the fact that they all were intact as secure loops of silk transfixing Cooper's ligament, but the loops were empty, and the edge of the conjoined tendon was about 1 inch away. The tantalum gauze previously implanted, however, was firmly attached to the original conjoined tendon and was so heavily infiltrated and surrounded by fibrous tissue as to make a strong downward, medial and lateral extension of that structure. The fibrous layer was approximately ⅛ inch thick and very strong. After properly

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dealing with the sac, the fibrous tissue infiltrated tantalum gauze was sutured to Cooper's ligament without any tension whatsoever. This made a very strong closure, and gave us a sense of security which we did not have at the previous operation. The remainder of the wound was closed as usual. Silk was used as suture material throughout.

To date (April 1, 1954) the patient has remained well.

COMMENT

This case, and the other one being reported elsewhere,[3] are unwitting examples of the use of tantalum gauze as a foundation for building a new fibrous structure to be used in the secondary repair of a hernia that has very poor tissues to start with. They may indeed be called two stage operations for hernia. It is believed that the reason the patient went three years after our first (her second) operation before she had a recurrence was due to the fact that the suture line was reinforced by a piece of tantalum gauze at the time of operation. We believe that the conjoined tendon pulled away from Cooper's ligament soon after operation. The fibrous tissue infiltrated tantalum gauze probably is what prevented the hernia from recurring very soon. If, at the previous operation, the edge of the tantalum gauze had been sutured to Cooper's ligament instead of to Poupart's, the chances are that the hernia would not have recurred. The tantalum probably would have remained in place when the conjoined tendon pulled away; would have become infiltrated with fibrous tissue, and would have formed an effective buttress between the abdominal cavity and the femoral canal. However, the tantalum gauze had been laid merely on top of the conjoined tendon-Cooper's ligament suture line and had not been sutured to the ligament.

SUMMARY

A case is reported of a recurrent femoral hernia with a poor conjoined tendon, which at operation was sutured to Cooper's ligament. A piece of tantalum gauze was laid over the suture line. Three years later the hernia recurred, the conjoined tendon having pulled completely away from Cooper's ligament. The overlying piece of tantalum gauze had become completely infiltrated with fibrous tissue, forming a new and ample shelf-like addition to the original conjoined tendon. This newly formed fibrous infiltrated tantalum gauze addition to the conjoined tendon was sutured to Cooper's ligament without tension, effecting a very firm closure.

REFERENCES

1. Halsted, W. S.: Cure of more difficult as well as simpler inguinal ruptures, Bull. Johns Hopkins Hosp. *14:* 208 (Aug.) 1903.
2. Koontz, A. R.: Tantalum gauze in hernia repair, Sinai Hosp. J. *1:* No. 2, 13 (Nov.) 1952.
3. Koontz, A. R.: Failures with tantalum gauze in ventral hernia repair. (To be published in Arch. Surg.).
4. Koontz, A. R.: Use of tantalum mesh in inguinal hernia repair, Surg., Gynec. & Obst. *92:* 101 (Jan.) 1951.

ON THE ANTISEPTIC PRINCIPLE IN THE PRACTICE OF SURGERY[1]

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In the course of an extended investigation into the nature of inflammation, and the healthy and morbid conditions of the blood in relation to it, I arrived, several years ago, at the conclusion that the essential cause of suppuration in wounds is decomposition, brought about by the influence of the atmosphere upon blood or serum retained within them, and, in the case of contused wounds, upon portions of tissue destroyed by the violence of injury.

To prevent the occurrence of suppuration, with all its attendant risks, was an object manifestly desirable; but till lately apparently unattainable, since it seemed hopeless to attempt to exclude the oxygen, which was universally regarded as the agent by which putrefaction was effected. But when it had been shown by the researches of Pasteur that the septic property of the atmosphere depended, not on the oxygen or any gasous consituent, but on minute organisms suspended in it, which owed their energy to their vitality, it occurred to me that decomposition in the injured part might be avoided without excluding the air, by applying as a dressing some material capable of destroying the life of the floating particles.

Upon this principle I have based a practice of which I will now attempt to give a short account.

The material which I have employed is carbolic or phenic acid, a volatile organic compound which appears to exercise a peculiarly destructive influence upon low forms of life, and hence is the most powerful antiseptic with which we are at present acquainted.

The first class of cases to which I applied it was that of compound fractures, in which the effects of decomposition in the injured part were especially striking and pernicious. The results might have been such as to establish conclusively the great principle, that *all the local inflammatory mischief and general febrile disturbance which follow severe injuries are due to the irritating and poisoning influence of decomposing blood or sloughs*. For these evils are entirely avoided by the antiseptic treatment, so that limbs which otherwise would be unhesitatingly condemned to amputation may be retained with confidence of the best results.

In conducting the treatment, the first object must be the destruction of any septic germs which may have been introduced into the wound, either at the moment of the accident or during the time which has since elapsed. This is done by introducing the acid of full strength into all accessible recesses of the wound

[1] A paper read before the British Medical Association in Dublin on the 9th of August, 1867.

by means of a piece of rag held in dressing forceps and dipped in the liquid.[2] This I did not venture to do in the earlier cases; but experience has shown that the compound which carbolic acid forms with the blood, and also any portions of tissue killed by its caustic action, including even parts of the bone, are disposed of by absorption and organisation, provided they are afterwards kept from decomposing. We are thus enabled to employ the antiseptic treatment efficiently at a period after the occurrence of the injury at which it would other wise probably fail. Thus I have now under my care in the Glasgow Infirmary a boy who was admitted with compound fracture of the leg as late as eight and a half hours after the accident, in whom nevertheless all local and constitutional disturbance was avoided by means of carbolic acid, and the bones were firmly united five weeks after his admission.

The next object to be kept in view is to guard effectually against the spreading of decomposition into the wound along (p. 354) the stream of blood and serum which oozes out during the first few days after the accident, when the acid originally applied has been washed out, or dissipated by absorption and evaporation. This part of the treatment has been greatly improved during the last few weeks. The method which I have hitherto published[3] consisted in the application of a piece of lint dipped in the acid, overlapping the sound skin to some extent, and covered with a tin cap, which was daily raised in order to touch the surface of the lint with the antiseptic. This method certainly succeeded well with wounds of moderate size; and, indeed, I may say that in all the many cases of this kind which have been so treated by myself or my house-surgeons, not a single failure has occurred. When, however, the wound is very large, the flow of blood and serum is so profuse, especially during the first twenty-four hours, that the antiseptic application cannot prevent the spread of decomposition into the interior unless it overlaps the sound skin for a very considerable distance, and this was inadmissible by the method described above, on account of the extensive sloughing of the surface of the cutis which it would involve. This difficulty has, however, been overcome by employing a paste composed of common whitening (carbonate of lime) mixed with a solution of one part of carbolic acid in four parts of boiled linseed oil, so as to form a firm putty. This application contains the acid in too dilute a form to excoriate the skin, which it may be made to cover to any extent that may be thought desirable, while its substance serves as a reservoir of the antiseptic material. So long as any discharge continues, the paste should be changed daily; and, in order to prevent the chance of mischief occurring during the process, a piece of rag dipped in the solution of carbolic aid in oil is put on next to the skin, and maintained there permanently, care being taken to avoid raising it along with the putty. This rag is always kept in an antiseptic condition from contact with the paste above it, and destroys any germs that may fall upon it during the short time that should alone be allowed to pass in the changing of the dressing. The putty should be in a layer about a quarter of an

[2] The addition of a few drops of water to a considerable quantity of the crystallised acid induces it to assume permanently the liquid form

[3] See The Lancet for March 16th, 23rd, and 30th, and April 27th, of the present year.

inch thick, and may be advantageously applied rolled out between two pieces of calico, which maintain it in the form of a continuous sheet, that may be wrapped in a moment round the whole circumference of a limb, if this be thought desirable while the putty is prevented by the calico from sticking to the rag which is next the skin.[4] When all discharge has ceased, the use of the paste is discontinued, but the original rag is left adhering to the skin till healing by scabbing is supposed to be complete. I have at present in the hospital a man with severe compound fracture of both bones of the left leg, caused by direct violence, who, after the cessation of the sanious discharge under the use of the paste, without a drop of pus appearing, has been treated for the last two weeks exactly as if the fracture were a simple one. During this time the rag, adhering by means of a crust of inspissated blood collected beneath it, has continued perfectly dry, and it will be left untouched till the usual period for removing the splints in a simple fracture, when we may fairly expect to find a sound cicatrix beneath it.

We cannot, however, always calculate on so perfect a result as this. More or less pus may appear after the lapse of the first week; and the larger the wound the more likely is this to happen. And here I would desire earnestly to enforce the necessity of persevering with the antiseptic application, in spite of the appearance of suppuration, so long as other symptoms are favourable. The surgeon is extremely apt to suppose that any suppuration is an indication that the antiseptic treatment has failed, and that poulticing or water-dressing should be resorted to. But such a course would in many cases sacrifice a limb or a life. I cannot, however, expect my professional breathren to follow my advice blindly in such a matter, and therefore I feel it necessary to place before them, as shortly as I can, some pathological principles, intimately connected not only with the point we are immediately considering, but with the whole subject of this paper.

If a perfectly healthy granulating sore be well washed and covered with a plate of clean metal, such as block tin, fitting its surface pretty accurately, and overlapping the surrounding skin an inch or so in every direction, and retained in position by adhesive plaster and a bandage, it will be found, on removing it after twenty-four or forty-eight hours, that little or nothing than can be called pus is present, merely a little transparent fluid, while at the same time there is an entire absence of the unpleasant odour invariably perceived when water-dressing is changed. Here the clean metallic surface presenting no recesses, like those of porous lint, for the septic germs to develop in, the fluid exuding from the surface of the granulations has flowed away undecomposed, and the result is absence of suppuration. This simple experiment illustrates the important fact, that granulations have no inherent tendency to form pus, but do so only when subjected to a preternatural stimulus. Further, it shows that the mere contact of a foreign body does not of itself stimulate granulations to suppurate; whereas the presence of decomposing organic matter does. These truths are even more strikingly exemplified by the fact, which I have elsewhere recorded,[5] that a piece of dead

[4] In order to prevent evaporation of the acid, which passes readily through any organic tissue, such as oiled silk or gutta percha, it is well to cover the paste with a sheet of block tin, or tinfoil strengthened with adhesive plaster. The thin sheet-lead used for lining tea-chests will also answer the purpose, and may be obtained from any wholesale grocer.

bone, free from decomposition, may not only fail to induce the granulations around it to suppurate, but may actually be absorbed by them; whereas a bit of dead bone soaked with putrid pus infallibly induces suppuration in its vicinty.

Another instructive experiment is to dress a granulating sore with some of the putty above described, overlapping the sound skin extensively, when we find in the course of twenty-four hours that pus has been produced by the sore, although the application has been perfectly antiseptic; and indeed, the larger the amount of carbolic acid in the paste the greater is the quantity of pus formed, provided we avoid such a proportion as would act as a caustic. The carbolic acid, though it prevents decomposition, induces suppuration—obviously by acting as a chemical stimulus; and we may safely infer that putrescent organic materials which we know to be chemically acrid) operate in the same way.

In so far, then, carbolic acid and decomposing substances are alike—namely, that they induce suppuration by chemical stimulation, as distinguished from what may be termed simple inflammatory suppuration, such as that in which ordinary abscesses originate, where the pus appears to be formed in consequence of an excited action of the nerves, independently of any other stimulus. There is, however, this enormous difference between the effects of carbolic acid and those of decomposition—viz., that carbolic acid stimulates only the surface to which it is first applied, and every drop of discharge that forms weakens the stimulant by diluting it. But decomposition is a self-propagating and self-aggravating poison; and if it occurs at the surface of a severely injured limb, it will spread into all its recesses so far as any extravasated blood or shreds of dead tissue may extend, and, lying in these recesses, it will become from hour to hour more acrid till it acquires the energy of a caustic, sufficient to destroy the vitality of any tissues naturally weak from inferior vascular supply, or weakened by the injury they sustained in the accident.

Hence it is easy to understand how, when a wound is very large, the crust beneath the rag may prove here and there insufficient to protect the raw surface from the stimulating influence of the carbolic acid in the putty, and the result will be, first, the conversion of the tissues so acted on into granulations, and subsequently the formation of more or less pus. This, however, will be merely superficial, and will not interfere with the absorption and organisation of extravasated blood or dead tissues in the interior; but, on the other hand, should decomposition set in before the internal parts have become securely consolidated, the most disastrous results may ensue.

I left behind me in Glasgow a boy, thirteen years of age, who between three and four weeks previously met with a most severe injury to the left arm, which he got entangled in a machine at a fair. There was a wound six inches long and three inches broad, and the skin was very extensively undermined beyond its limits, while the soft parts generally were so much lacerated that a pair of dressing forceps introduced at the wound, and pushed directly inwards, appeared beneath the skin at the opposite aspect of the limb. From this wound several tags of muscle were hanging, and among them there was one consisting of about three inches of the triceps in almost its entire thickness; while the lower fragment of

[5] See The Lancet, March 23rd, 1867.

the bone, which was broken high up, was protruding four and a half inches, stripped of muscle, the skin being tucked in under it. Without the assistance of the antiseptic treatment, I should certainly have thought of nothing else but amputation at the shoulder-joint; but as the radial pulse could be felt, and the fingers had sensation, I did not hesitate to try to save the limb, and adopted the plan of treatment above described, wrapping the arm from the shoulder to below the elbow in the antiseptic application, the whole interior of the wound, together with the protruding bone, having previously been freely treated (p. 355) with strong carbolic acid. About the tenth day the discharge, which up to that time had been only sanious and serous, showed a slight admixture of slimy pus, and this increased till, a few days before I left, it amounted to about three drachms in twenty-four hours. But the boy continued, as he had been after the second day, free from unfavourable symptoms, with pulse, tongue, appetite, and sleep natural, and strength increasing, while the limb remained, as it had been from the first, free from swelling, redness, or pain. I therefore persevered with the antiseptic dressing, and before I left, the discharge was already somewhat less, while the bone was becoming firm. I think it likely that in that boy's case I should have found merely a superficial sore had I taken off all the dressings at the end of three weeks, though, considering the extent of the injury, I thought it prudent to let the month expire before disturbing the rag next to the skin. But I feel sure that if I had resorted to ordinary dressing when the pus first appeared, the progress of the case would have been exceedingly different.

The next class of cases to which I have applied the antiseptic treatment is that of abscesses. Here, also, the results have been extremely satisfactory, and in beautiful harmony with the pathological principles indicated above. The pyogenic membrane, like the granulations of a sore, which it resembles in nature, forms pus, not from any inherent disposition to do so, but only because it is subjected to some preternatural stimulation. In an ordinary abscess, whether acute or chronic, before it is opened, the stimulus which maintains the suppuration is derived from the presence of the pus pent up within the cavity. When a free opening is made in the ordinary way, this stimulus is got rid of; but the atmosphere gaining access to the contents, the potent stimulus of decomposition comes into operation, and pus is generated in greater abundance than before. But when the evacuation is effected on the antiseptic principle, the pyogenic membrane, freed from the influence of the former stimulus without the substitution of a new one, ceases to suppurate (like the granulations of a sore under metallic dressing), furnishing merely a trifling amount of clear serum, and, whether the opening be dependent or not, rapidly contracts and coalesces. At the same time any constitutional symptoms previously occasioned by the accumulation of the matter are got rid of without the slightest risk of the irritative fever or hectic hitherto so justly dreaded in dealing with large abscesses.

In order that the treatment may be satisfactory, the abscess must be seen before it has opened. Then, except in very rare and peculiar cases,[6] there are no

[6] As an instance of one of these exceptional cases, I may mention that of an abscess in the vicinity of the colon, and afterwards proved by post-mortem examination to have once communicated with it. Here the pus was extremely offensive when evacuated, and exhibited vibrios under the microscope.

septic organisms in the contents, so that it is needless to introduce carbolic acid into the interior. Indeed, such a proceeding would be objectionable, as it would stimulate the pyogenic membrane to unnecessary suppuration. All that is necessary is to guard against the introduction of living atmospheric germs from without, at the same time that free opportunity is afforded for the escape of discharge from within.

I have so lately given elsewhere[7] a detailed account of the method by which this is effected, that it is needless for me to enter into it at present, further than to say that the means employed are the same as those described above for the superficial dressing of compound fractures—namely, a piece of rag dipped in the solution of carbolic acid in oil, to serve as an antiseptic curtain, under cover of which the abscess is evacuated by free incision; and the antiseptic paste, to guard against decomposition occurring in the stream of pus that flows out beneath it: the dressing being changed daily till the sinus has closed.

The most remarkable results of this practice in a pathological point of view have been afforded by cases where the formation of pus depended upon disease of bone. Here the abscesses, instead of forming exceptions to the general class in the obstinacy of the suppuration, have resembled the rest in yielding in a few days only a trifling discharge; and frequently the production of pus has ceased from the moment of the evacuation of the original contents. Hence it appears that caries, when no longer labouring, as heretofore, under the irritation of decomposing matter, ceases to be an opprobrium of surgery, and recovers like other inflammatory affections. In the publication before alluded to[8] I have mentioned the case of a middle-aged man with psoas abscess depending on diseased bone, in whom the sinus finally closed after months of patient perseverance with the antiseptic treatment. Since that article was written, I have had another instance of success, equally gratifying, but differing in the circumstance that the disease and the recovery were both more rapid in their course. The patient was a blacksmith who had suffered four and a half months before I saw him from symptoms of ulceration of cartilage in the left elbow. These had latterly increased in severity, so as to deprive him entirely of his night's rest and of appetite. I found the region of the elbow greatly swollen, and on careful examination discovered a fluctuating point at the outer aspect of the articulation. I opened it on the antiseptic principle, the incision evidently penetrating to the joint, giving exit to a few drachms of pus. The medical gentleman under whose care he was (Dr. Macgregor of Glasgow) supervised the daily dressing with the carbolic-acid paste till the patient went to spend two or three weeks at the coast, when his wife was entrusted with it. Just two months after I opened the abscess he called to show me the limb, stating that the discharge had for at least two weeks been as little as it then was —a trifling moisture upon the paste, such as might be accounted for by the little sore caused by the incision. On applying a probe guarded with an antiseptic rag, I found that the sinus was soundly closed, while the limb was free from swelling or tenderness; and, although he had not attempted to exercise it much, the joint

[7] See The Lancet of July 27th, 1867.
[8] Ibid.

could already be moved through a considerable angle. Here the antiseptic principle had effected the restoration of a joint which on any other known system of treatment must have been excised.

Ordinary contused wounds are of course amenable to the same treatment as compound fractures, which are a complicated variety of them. I will content myself with mentioning a single instance of this class of cases. In April last a volunteer was discharging a rifle, when it burst, and blew back the thumb with its metacarpal bone, so that it could be bent back as on a hinge at the trapezial joint, which had evidently been opened, while all the soft parts between the metacarpal bones of the thumb and forefinger were torn through. I need not insist before my present audience on the ugly character of such an injury. My house-surgeon, Mr. Hector Cameron, applied carbolic acid to the whole raw surface, and completed the dressing as if for compound fracture. The hand remained free from pain, redness, or swelling, and, with the exception of a shallow groove, all the wound consolidated without a drop of matter, so that if it had been a clean cut, it would have been regarded as a good example of primary union. The small granulating surface soon healed, and at present a linear cicatrix alone tells of the injury he sustained, while his thumb has all its movements and his hand a firm grasp.

If the severest form of contused and lacerated wounds heal thus kindly under the antiseptic treatment, it is obvious that its application to simple incised wounds must be merely a matter of detail. I have devoted a good deal of attention to this class, but I have not as yet pleased myself altogether with any of the methods I have employed. I am, however, prepared to go so far as to say that a solution of carbolic acid in twenty parts of water, while a mild and cleanly application, may be relied on for destroying any septic germs that may fall upon the wound during the performance of an operation; and also that for preventing the subsequent introduction of others, the paste above described, applied as for compound fractures, gives excellent results. Thus I have had a case of strangulated inguinal hernia, in which it was necessary to take away half a pound of thickened omentum, heal without any deep-seated suppuration or any tenderness of the sac or any fever; and amputations, including one immediately below the knee, have remained absolutely free from constitutional symptoms.

Further, I have found that when the antiseptic treatment is efficiently conducted, ligatures may be safely cut short and left to be disposed of by absorption or otherwise. Should this particular branch of the subject yield all that it promises, should it turn out on further trial that when the knot is applied on the antiseptic principle, we may calculate as securely as if it were absent on the occurrence of healing without any deep-seated suppuration; the deligation of main arteries in their continuity will be deprived of the two dangers that now attend it—viz., those of secondary hemorrhage and an unhealthy state of the wound. Further, it seems not unlikely that the present objection to tying an artery in the immediate vicinity of a large branch may be done away with; and that even the innominate, which has lately been the subject of an ingenious experiment by one of the Dublin surgeons on account of its well-known fatality under the ligature

from secondary hemorrhage, may cease to have this unhappy character, when the tissues in the vicinity of the thread, instead of becoming softened through the influence of an irritating decomposing (p. 356) substance, are left at liberty to consolidate firmly near an unoffending though foreign body.

It would carry me far beyond the limited time which, by the rules of the Association, is alone at my disposal, were I to enter into the various applications of the antiseptic principle in the several special departments of surgery.

There is, however, one point more that I cannot but advert to—namely, the influence of this mode of treatment upon the general healthiness of a hospital. Previously to its introduction, the two large wards in which most of my cases of accident and of operation are treated were amongst the unhealthiest in the whole surgical division of the Glasgow Royal Infirmary, in consequence, apparently, of these wards being unfavourably placed with reference to the supply of fresh air; and I have felt ashamed, when recording the results of my practice, to have so often to allude to hospital gangrene or pyaemia. It was interesting, though melancholy, to observe that, whenever all, or nearly all, the beds contained cases with open sores, these grievous complications were pretty sure to show themselves; so that I came to welcome simple fractures, though in themselves of little interest either for myself or the students, because their presence diminished the proportion of open sores among the patients. But since the antiseptic treatment has been brought into full operation, and wounds and abscesses no longer poison the atmosphere with putrid exhalations, my wards, though in other respects under precisely the same circumstances as before, have completely changed their character; so that during the last nine months not a single instance of pyaemia, hospital gangrene, or erysipelas has occurred in them.

As there appears to be no doubt regarding the cause of this change, the importance of the fact can hardly be exaggerated.

BOOK REVIEWS

The editors of THE AMERICAN SURGEON will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.

Preservation and Transplantation of Normal Tissues. Editors for the Ciba Foundations; G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Margaret P. Cameron, M.A., A.B.L.S., Assisted by Joan Etherington, With 55 Illustrations; Little, Brown, and Company. Boston, 1954. Price $6.50.

Anyone interested in the preservation and transplantation of normal tissues will find this symposium a valuable volume. A basic discussion of the immunity problem is ably reviewed. The sections on blood vessel, skin, bone, and corneal transplants are of significant clinical application. Methods of preparation and storage of various tissues are discussed. The preservation of tissue banks is of practical value for anyone contemplating the establishment of such a service. The biophysical aspect of freezing living cells as a method of preservation is reviewed. This collection of basic and clinical observations by the leading workers in this field is a timely and very worthwhile addition.

CREIGHTON A. HARDIN, M.D.

BOOKS RECEIVED

Books received are acknowledged in this section, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

The Bacterial Factor in Traumatic Shock. By; Jacob Fine, M.D., Department of Surgery, Beth Israel Hospital, Harvard Medical School, Boston, Massachusetts. Charles C Thomas, Publisher, Springfield, Illinois, U. S. A., 1954. Price $2.75.

Peripheral Circulation in Man. Editors for the Ciba Foundation. G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch. and Jessie S. Freeman, M.B., B.S., D.P.H., Assisted by Joan Etherington, With 72 Illustrations. Little, Brown and Company, Boston, 1954. Price $6.00.